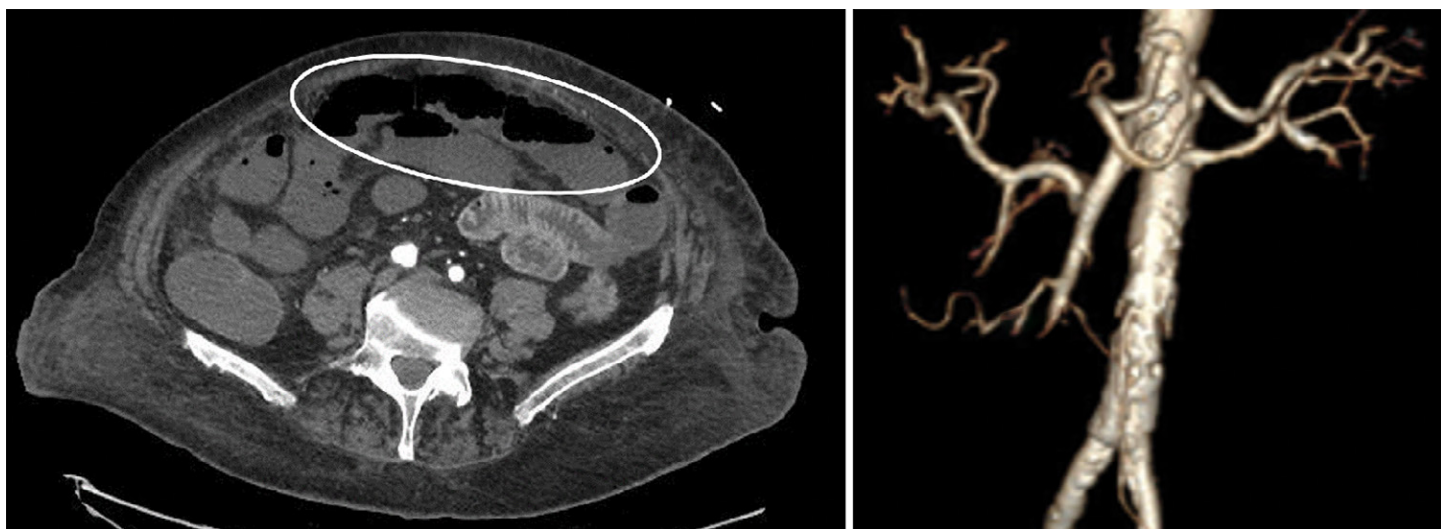


Acute Mesenteric Ischemia: Pathophysiology-based Approach to Imaging Findings and Diagnosis

Matthew H. Lee, MD¹ • Perry J. Pickhardt, MD¹ • Anna M. Sorensen, MD¹ • Giuseppe V. Toia, MD, MS¹ • Meghan G. Lubner, MD¹
Ryan Sappenfield, MD² • David H. Kim, MD¹

Author affiliations, funding, and conflicts of interest are listed at [the end of this article](#).
See the invited commentary by [Huete](#) in this issue.



Acute mesenteric ischemia (AMI) is an abdominal emergency characterized by a sudden decrease in blood flow to meet the metabolic demands of bowel. AMI is uncommon but associated with high morbidity and mortality. Imaging plays a decisive role in early diagnosis and management because the symptoms of AMI are often nonspecific without specific laboratory tests or serologic biomarkers for early diagnosis. AMI comprises distinct entities with unique pathophysiology, imaging features, and management strategies. The basic causal mechanisms underlying AMI are inadequate inflow (arterial occlusion), inadequate outflow (mesenteric venous occlusion), global hypoperfusion (nonobstructive mesenteric ischemia [NOMI]), and strangulating bowel obstruction. Identifying transmural necrosis is critical for all causes of AMI. The authors review the foundational anatomy and pathophysiology of AMI, its distinct imaging features, and a systematic approach to AMI with emphasis on up-to-date imaging findings predictive of transmural necrosis. They emphasize a pathophysiology-based approach to AMI rather than a semiology-based approach (ie, patterns and signs) because the imaging features, significance, and predictive value of the imaging findings vary based on the underlying cause of AMI, with arterial occlusive AMI and NOMI having worse prognosis than mesenteric venous AMI. The authors highlight the specific vascular, bowel, and extraintestinal findings for each type of AMI with emphasis on imaging predictors of transmural bowel necrosis and address points of confusion to equip interpreting radiologists with a foundational understanding of AMI.

©RSNA, 2025 • radiographics.rsna.org

SHOUT
SHARE IT





Supplemental
Material

Test Your
Knowledge



RadioGraphics 2025; 45(10):e250012
<https://doi.org/10.1148/rg.250012>

Content Codes: CT, ER, GI, IR

Abbreviations: AMI = acute mesenteric ischemia, CLO = closed-loop obstruction, MVT = mesenteric venous thrombosis, NOMI = nonocclusive mesenteric ischemia, OR = odds ratio, PVP = portal venous phase, SBO = small-bowel obstruction, SMA = superior mesenteric artery, SMV = superior mesenteric vein

TEACHING POINTS

- There are no specific laboratory measures or serum biomarkers for diagnosing early, potentially reversible AMI in routine clinical practice. Levels of conventional serologic markers such as serum lactate are elevated late in the disease course after transmural infarction and the serum lactate level is often normal early, which may be misleading and harmful because normal lactate level is an independent predictor of delayed AMI diagnosis.
- Imaging is critically important for diagnosing AMI and evaluating the extent of end-organ injury and prognosis, and CT angiography is the imaging study of choice for suspected AMI according to the American College of Radiology, Society for Vascular Surgery, and European Society for Vascular Surgery.
- The severity of arterial vascular narrowing (ie, >70% stenosis, complete occlusion) and pneumatosis intestinalis are predictors of transmural necrosis. Bowel dilatation and extreme bowel wall thinning with an imperceptible paper-thin appearance are predictive of transmural necrosis and related to irreversible injury to the deep muscle layers and myenteric plexus. Wall thinning is 38% sensitive and 98% specific for transmural necrosis. Bowel dilatation is more common in arterial occlusion than in venous occlusion or NOMI; importantly, bowel dilatation should be recognized as a predictor of transmural bowel necrosis in arterial occlusive AMI, not misinterpreted as simple ileus.
- Decreased or absent bowel wall enhancement (diagnostic odds ratio [DOR] = 8.2, $P = .00$) and bowel dilatation (DOR = 3.1, $P = .00$) are statistically significant predictors of transmural necrosis in venous AMI. Decreased or absent wall enhancement reflects cessation of arterial flow related to later-stage ischemia. Importantly, the degree of wall thickening and extraintestinal findings do not correlate with the severity of ischemic bowel injury.
- In patients with NOMI, CT features associated with transmural necrosis were absent wall enhancement ($P < .001$), wall thinning ($P < .001$), bowel dilatation ($P = .041$), pneumatosis ($P = .003$), mesenteric gas ($P = .005$), and portal venous gas ($P = .002$). Using clinical and imaging findings associated with irreversible transmural necrosis, Calame et al identified four predictive factors—absent bowel enhancement, bowel thinning, plasma bicarbonate concentration of 15 mmol/L or less, and prothrombin rate less than 40%—to devise a system for risk stratification whereby 88% and 100% of patients with three or four of these features, respectively, had irreversible transmural bowel necrosis.

Introduction

Acute mesenteric ischemia (AMI) is an uncommon but life-threatening abdominal emergency characterized by inability of blood flow to meet the metabolic demands of the bowel and visceral organs. The abrupt decrease in blood supply seen with AMI results in cellular damage, bowel necrosis, and death, if untreated (1). Although AMI is infrequent, accounting for fewer than one in 1000 hospital admissions, mortality remains high (50%–80%) (1–4), with devastating consequences if unrecognized and undiagnosed. As with other forms of end-organ ischemia, time is of the essence, and prompt recognition, diagnosis, and treatment are par-

amount. Unfortunately, early diagnosis is difficult owing to the low prevalence and often nonspecific clinical manifestation and laboratory findings. Indeed, AMI is a high-risk and low-prevalence disease that requires a high level of clinical suspicion (5). CT plays a vital role in diagnosing AMI and complications thereof, and radiologists who interpret abdominal CT should be comfortable with the basic pathophysiology, imaging findings, and associated prognosis.

AMI is not a single entity but rather multiple distinct entities that can be distinguished by their pathophysiology, clinical manifestation, imaging features, and prognosis (6). Although each entity can result in irreversible transmural bowel necrosis, the vascular, bowel, and extraintestinal imaging findings are not generalizable. Rather, the findings need to be contextualized by the underlying pathophysiology. Although imaging signs are important, confusion about AMI abounds in part because of the rarity of AMI but also because these entities have been comingled and imaging features have at times been conflated (7).

The purpose of this review is to provide a contemporary evidence-based appraisal of AMI with an emphasis on the role of imaging in establishing the diagnosis and prognosis. The authors present a pathophysiology-based interpretive approach to AMI that highlights the specific vascular, bowel, and extraintestinal findings for each form of AMI and the imaging predictors of transmural bowel necrosis, which differ by cause (Table S1) (8). Foundational concepts and the role of imaging are reviewed for each entity, with an emphasis on up-to-date imaging features associated with transmural infarction and areas of ongoing confusion and controversy.

Foundational Concepts: Anatomy and Pathophysiology

Mesenteric ischemia occurs when blood flow is insufficient to meet the metabolic demands of bowel. The causal mechanisms of AMI can be distilled into four basic underlying causes: inadequate inflow (eg, arterial occlusion), inadequate outflow (eg, mesenteric venous occlusion), global hypoperfusion (eg, nonocclusive mesenteric ischemia [NOMI]), and strangulating bowel obstruction (9,10). Practically speaking, the first three flow-related causes are primary causes of AMI, whereas strangulating bowel obstruction can be thought of as secondary AMI (Fig 1). A confident understanding of the relationships between the arterial and mesenteric vasculature, vascular territories, and bowel is foundational to understanding the pathophysiology and imaging features of the different forms of AMI (Fig 1).

Vascular Anatomy and Vascular Territories

The mesenteric circulation is an exquisite interconnected network where collaterals and redundancy are meant to ensure against the devastating consequences that would result from loss of a single vessel (2). The celiac artery, superior mesenteric artery (SMA), and inferior mesenteric artery (IMA) are the three primary aortic arterial branches supplying the visceral organs, with several important collateral pathways pertinent to the pathophysiology of bowel ischemia (Fig 2) (Table). The SMA is the dominant arterial supply to the midgut and small

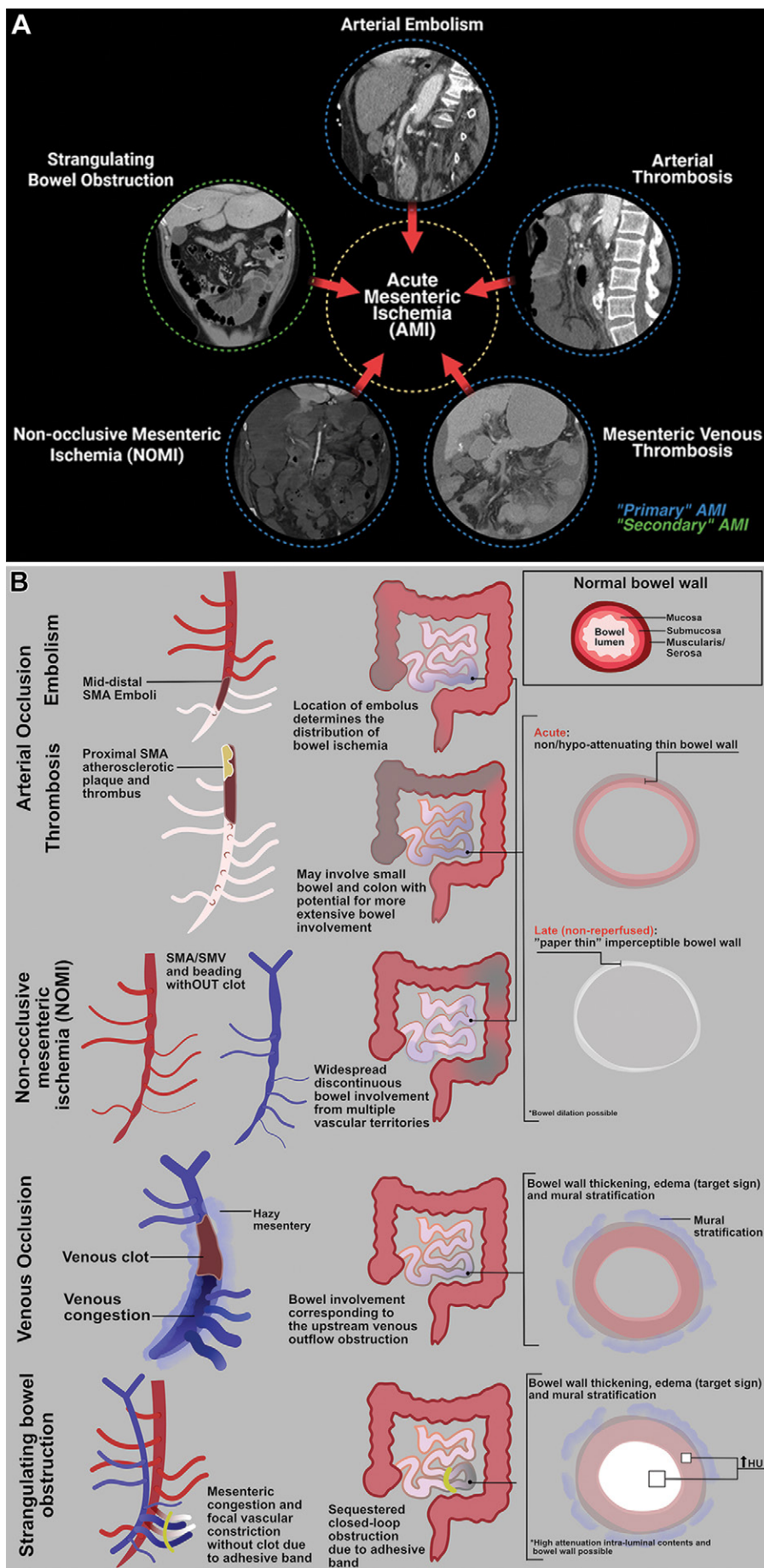


Figure 1. Different causes of AMI. **(A)** The causes can be distilled into four basic underlying causes: inadequate inflow (eg, arterial occlusion; embolism and thrombosis), inadequate outflow (eg, mesenteric venous occlusion), global hypoperfusion (eg, NOMI), and strangulating bowel obstruction. (Created with BioRender.com; M. Lee, 2025; <https://BioRender.com/zo4o4kg>.) **(B)** Illustration shows a visual summary of AMI by cause, highlighting the characteristic vascular and bowel findings. SMA = superior mesenteric artery, SMV = superior mesenteric vein. (Illustration by A.M.S.)

bowel—with some collateral circulation from the celiac trunk and inferior mesenteric artery (9)—and should be regarded as any critical arterial supply to vital end-organs, analogous to the cerebral and coronary arteries for stroke and myocardial infarction, respectively. Despite theoretical fail-safes afforded by the network of arterial collaterals, collaterals typically develop incrementally with worsening stenosis over time, as can be seen in atherosclerosis or chronic vasculitis, but are not well developed to compensate in acute SMA occlusion. Given the large and critical territory supplied by the SMA, acute uncompensated occlusion can result in devastating intestinal ischemia.

The mesenteric venous anatomy parallels the arterial anatomy and drains the bowel supplied by the arteries (Fig 2) (11). The main draining vein for the small bowel, ascending colon, and transverse colon is the superior mesenteric vein (SMV) via the jejunal, ileal, right colic, and middle colic veins (9,12). The descending and sigmoid colon drain into the inferior mesenteric vein (IMV). The IMV then empties into the splenic vein, which joins the SMV to form the main portal vein, which delivers blood to the liver.

Pathophysiology of Bowel Ischemia

Bowel is metabolically demanding, with the normal bowel receiving 20%–25% of cardiac output. Approximately 33% of the total blood volume goes to the small bowel at rest, making it the largest reservoir for the circulatory system. The small intestine is particularly susceptible to ischemia, with the majority of blood flow being directed to the mucosa (70%–85%), followed by the serosa and muscularis (15%–20%) (9,13). The mucosa has the highest metabolic demand and is at greatest risk for ischemic injury, with mucosal injury occurring after 20–60 minutes of reduced blood flow. This can progress to transmural infarction within 8–12 hours (9). The severity and reversibility of bowel injury is related to the location of vascular occlusion or obstruction (if present), extent of reduced blood flow, number of vessels involved, presence of collateral vessels, and duration of ischemia. Although collateral circulation can be protective for up to 12 hours when as much as 75% of bowel perfusion is lost, significant bowel injury ensues after just 4–6 hours of complete loss of perfusion (14).

Ischemia incites an inflammatory response, resulting in vascular permeability and wall breakdown. Acutely, bowel typically appears dusky (dark-red brown), hemorrhagic, or thickened and edematous if the injury extends into the underlying submucosa and muscularis propria (Fig 3). Classically, there is sharp demarcation between infarcted and uninvolved bowel. Early histologic features of ischemia include mucin loss, hemorrhagic necrosis of the mucosa, lamina propria congestion and hyalinization, and atrophic crypts. The submucosa and muscularis propria are usually affected only in severe ischemia and may appear edematous or hemorrhagic. Transmural infarct can lead to perforation and peritonitis. Significant inflammation is typically absent acutely; however, neutrophils may be seen after reperfusion. Ischemia reperfusion injury is the result of restoring blood flow to injured bowel: neutrophil-mediated inflammation and oxidative stress that further injures bowel, leading to cell death (7,15).

Ischemic colitis (also known as colon ischemia) is a related but distinct entity resulting from transient local nonocclusive hypoperfusion, ischemia, and reperfusion injury (16) and is different from colon ischemia seen with AMI. A segmental pattern of colonic involvement is typical, with the left colon being most commonly affected, followed by the right colon and entire colon (16). Watershed zones at the junction of vascular supplies are particularly susceptible (Table). Although ischemic colitis is not the focus of the present work, isolated right-sided ischemic colitis is associated with worse clinical outcomes, higher mortality, and AMI (7,16).

Clinical Manifestation and Evaluation and Treatment Overview

Classically, AMI manifests with “pain out of proportion to the clinical examination,” related to the progression of bowel injury from the mucosa outward to the serosa (1). Subjective pain can be intense, with the initial mucosal injury characterized by sudden-onset cramping abdominal pain. During early ischemic injury, the bowel wall layers mask the pain or tenderness elicited with palpation. Pain increases with transmural involvement, and only late in the disease process do the examination findings match the severity of the bowel injury. Most commonly, patients present with nonspecific nonlocalized abdominal pain, nausea, vomiting, bloating, and diarrhea with occasional bloody stool (17). Late findings include peritonitis and sepsis related to transmural necrosis, perforation, or secondary reactive inflammation (18), which can progress to multisystem organ failure.

There are no specific laboratory measures or serum biomarkers for diagnosing early, potentially reversible AMI in routine clinical practice (7,18–20). Levels of conventional serologic markers such as serum lactate are elevated late in the disease course after transmural infarction (19) and the serum lactate level is often normal early, which may be misleading and harmful because normal lactate level is an independent predictor of delayed AMI diagnosis (20). D-lactate, ischemia-modified albumin, and intestinal fatty-acid binding protein (I-FABP) are promising newer biomarkers for diagnosis; however, there is no biomarker for early diagnosis that is rapid, stable, highly sensitive and specific, inexpensive, and easily measured, to our knowledge (21). Although laboratory measures are nonspecific for early diagnosis, they are predictive of necrosis and useful for guiding treatment.

Early recognition and timely surgical and endovascular intervention are the cornerstones of AMI management. AMI mortality is lowest when intervention occurs within 12 hours of symptom onset (22), with dramatic increases in mortality after 24 hours; less than 30% of patients survive when diagnosed more than 24 hours from symptom onset (23). Prompt intervention confers mortality benefit, and mortality increases from 11% when treated within 24 hours to 73% when treated after 24 hours (23). As with any form of end-organ ischemia, but specifically with impaired arterial inflow, there is time-dependent ischemic bowel injury that can progress to transmural necrosis, organ failure, and death. Distinguishing reversible ischemia from irreversible transmural necrosis is essential, because patients with the latter will need to

Important Arterial Vascular Anatomy in AMI			
Artery	Major Branches	Structures Supplied	Collateral Pathways
Celiac	Left gastric	Stomach	Celiac and SMA: Pancreaticoduodenal arcades (anterior and posterior) Dorsal pancreatic artery pathways Arc of Buhler Arc of Barkow
	Splenic	Spleen	
	Common hepatic	Liver, pancreas	
	Gastroduodenal (GDA)		
	Proper hepatic		
SMA	Right and left hepatic		SMA and IMA: Artery of Drummond Arc of Riolan Watershed zones: Splenic flexure (Griffith point): junction of middle colic (SMA) and left colic (IMA) Rectosigmoid junction (Sudeck point): junction of superior rectal artery (IMA) and middle or inferior rectal arteries
	Inferior pancreaticoduodenal (IPDA)	Duodenum, pancreas	
	Jejunal	Jejunum	
	Ileal	Ileum	
	Ileocolic	Distal ileum, cecum, appendix, ascending colon	
IMA	Right colic	Ascending colon	
	Middle colic	Ascending colon, proximal two-thirds of transverse colon	
	Left colic	Distal one-third of transverse colon, descending colon	
	Sigmoidal	Sigmoid colon	
	Superior rectal	Rectum	

* IMA = inferior mesenteric artery.

undergo surgical exploration with bowel resection of nonviable bowel. There are also statistically significant differences in mortality based on the underlying cause, between patients with transmural necrosis from arterial (74%) and mesenteric venous (42%) AMI (odds ratio [OR] = 3.5, $P < .001$) and those with transmural necrosis from NOMI (69%) and mesenteric venous AMI (44%) (OR = 3.2, $P < .001$) (24).

Treatment paradigms for AMI depend on the underlying cause and severity of bowel ischemia, requiring a multidisciplinary approach (1) (Fig S1). Although beyond the scope of this review, the treatment aims in AMI are to (a) restore compromised blood flow to the bowel and (b) resect nonviable segments of bowel while preserving as much functional bowel as possible (18). Typically, arterial embolism or thrombosis is treated with endovascular or open thrombectomy (14); NOMI with supportive care, catheter-directed mesenteric artery papaverine injection, and anticoagulation (18); mesenteric venous thrombosis with anticoagulation (14); and strangulated bowel obstruction with operative management and resection of nonviable bowel (25).

Imaging Paradigms for AMI

Imaging is critically important for diagnosing AMI and evaluating the extent of end-organ injury and prognosis, and CT angiography is the imaging study of choice for suspected AMI according to the American College of Radiology, Society for Vascular Surgery, and European Society for Vascular Surgery

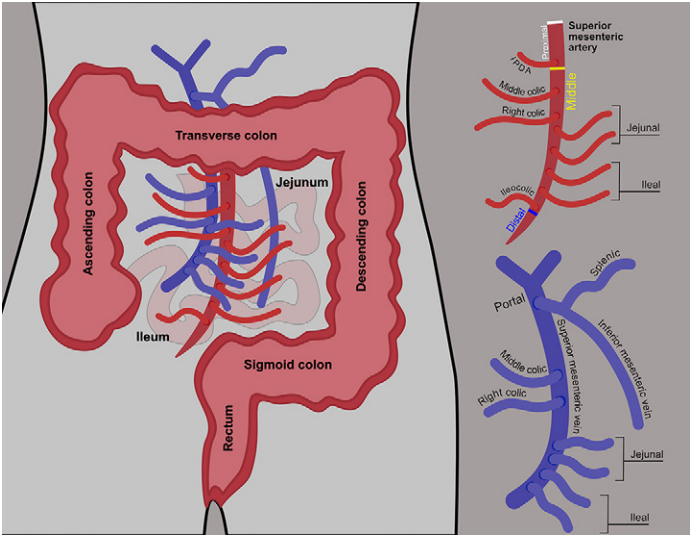


Figure 2. Normal vascular anatomy. The arterial image (top right) shows a practical standardized segmentation dividing the SMA into three segments: proximal SMA (S1): SMA origin to the origin of the inferior pancreaticoduodenal artery (IPDA), which is an important foregut collateral; middle SMA (S2): between the origin of the IPDA and the origin of the ileocolic artery (this segment includes the jejunal, right colic, and middle colic arteries); and distal SMA (S3): distal to the ileocolic artery. (Illustration by A.M.S.)

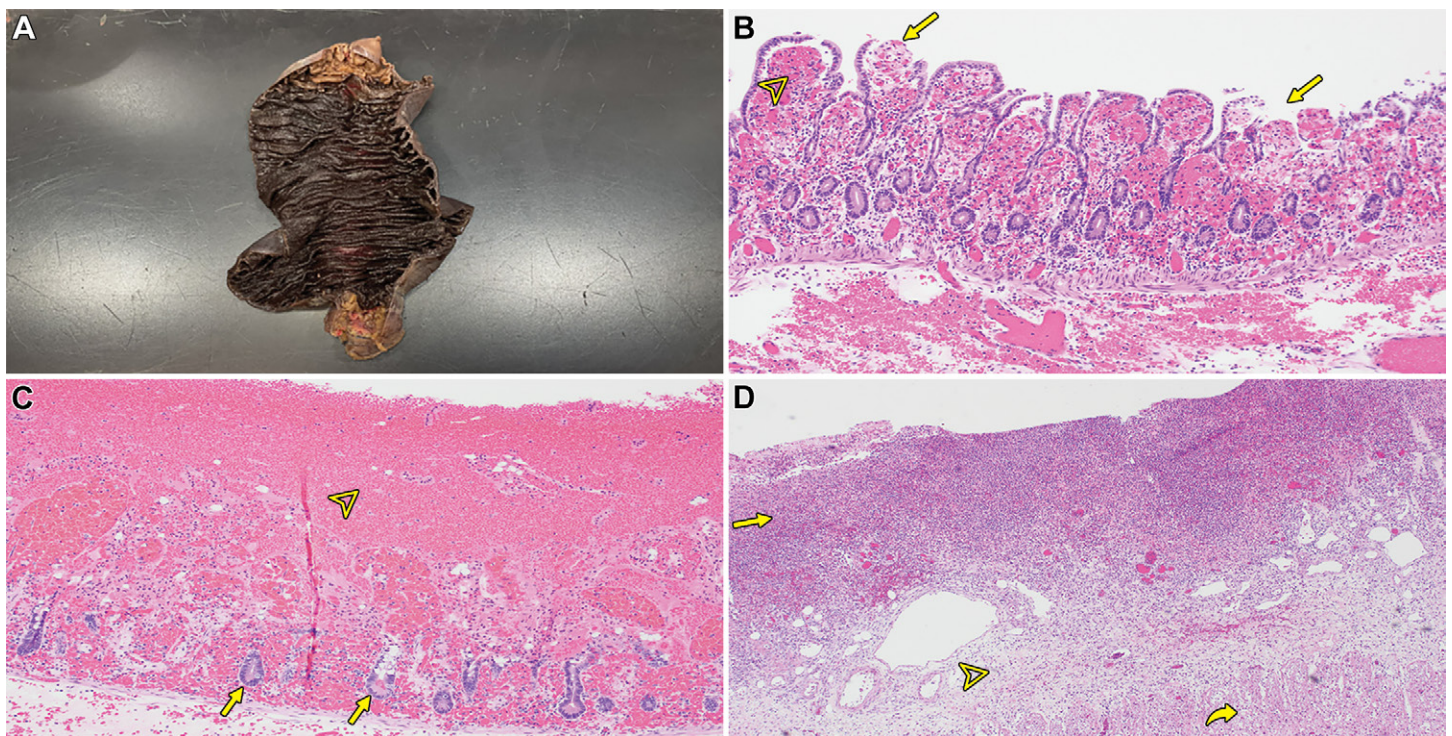


Figure 3. Gross and microscopic (histologic) examples of ischemic bowel disease. **(A)** Photograph from small-bowel resection shows dusky hemorrhagic mucosa. **(B, C)** Photomicrographs of early ischemic changes show superficial mucosal damage (arrows in **B**), congestion (arrowhead in **B**), hemorrhagic epithelial necrosis (arrowhead in **C**), and focal crypt preservation (arrows in **C**). (Hematoxylin-eosin [H-E] stain; original magnification, $\times 40$ [**B**] and $\times 100$ [**C**].) **(D)** Photomicrograph shows transmural infarction resulting from ischemia, which is typically due to acute vascular obstruction. Straight arrow = mucosa, arrowhead = submucosa, curved arrow = muscularis propria. (H-E stain; original magnification, $\times 20$.)

(26). A dedicated multiphase CT angiography mesenteric ischemia protocol is an integral part of the diagnostic algorithm because it is rapid, accessible, widely available, and highly effective, with sensitivity and specificity for diagnosing AMI between 90% and 100% (26–28). Multiphasic CT angiography enables direct visualization of the mesenteric arterial and venous vasculature and assessment of the extent and severity of bowel injury for treatment decision making (27) and allows detection of alternative diagnoses. Although efforts have been made to stage bowel ischemia with imaging (29), distinguishing between early, intermediate, and late ischemia may not always be feasible or clinically meaningful. Rather, understanding the imaging features of transmural necrosis is the most clinically impactful.

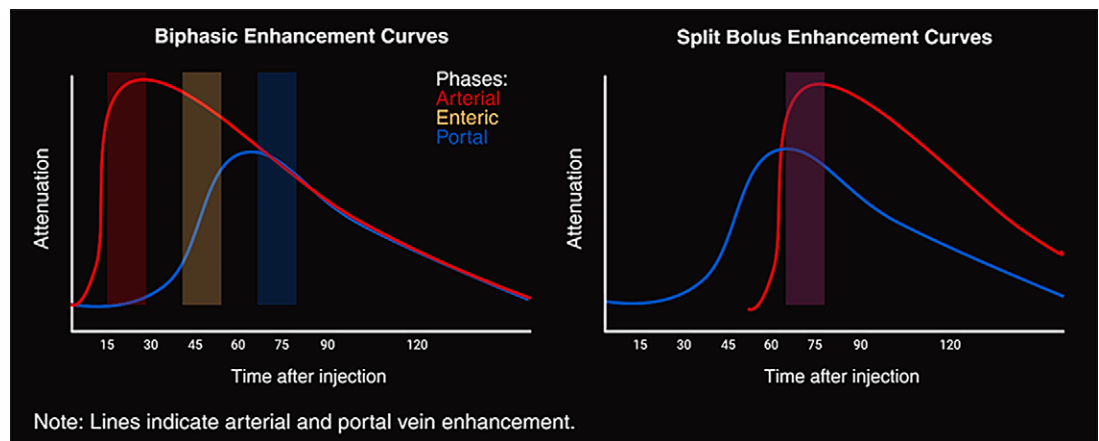
Multiphase CT angiography including noncontrast, arterial, and portal venous phase (PVP) images remains the first-line recommended study for suspected AMI (7,26). Although an initial nonenhanced series has been advocated to identify intramural hemorrhage associated with bowel ischemia and as a baseline to improve assessment of the bowel wall for absent or decreased enhancement (27,30), the necessity has been challenged (11,31). Rather, normal enhancing loops of bowel can be an internal reference from which to determine abnormal bowel enhancement, which can be assessed on PVP images (31). A single acquisition using a split-bolus technique—in which two contrast material boluses are administered with a set timed delay and single-phase acquisition (Fig 4)—can be considered because it captures the time when

arterial and PVP enhancement are greatest, allowing optimal assessment of the vasculature and bowel. This enables accurate and confident diagnosis while decreasing patient radiation and the number of images to review (32).

Strict prescription and adherence to a particular protocol cannot be considered practical or mandatory (7), as many patients will undergo PVP CT of the abdomen in the acute setting and PVP images are often the only images available. Importantly, the main features of AMI such as decreased bowel wall enhancement, bowel thinning and dilatation, and arterial occlusion can be identified on PVP images (7,31), although assessment for arterial lesions with PVP CT alone is associated with increased false-positives (31). Therefore, radiologists should be cognizant of the imaging features of AMI when routine PVP CT is performed in a patient with acute abdominal pain, knowing that AMI is underdiagnosed without a stated clinical suspicion and that an AMI diagnosis is statistically more likely when explicitly stated in the indication (33). Additionally, although neutral oral contrast material can improve bowel wall assessment (eg, thickness, enhancement), oral contrast material of any kind should typically be avoided when AMI is suspected, as it may not be tolerated and could result in unnecessary delays in care (28,34).

The improved tissue characterization and differentiation enabled with dual-energy CT (DECT) have clear application to AMI (35,36) (Fig S2). DECT allows identification of early bowel ischemia and significantly improves the conspicuity of hypoenhancing bowel wall compared with that of conventional

Figure 4. Theoretical enhancement curves with example mesenteric ischemia protocols. Left: Biphasic protocols have dedicated separately acquired arterial and portal venous phases. Right: Split-bolus protocols use two contrast material boluses separated by a set timed delay with a single-phase acquisition, to best represent a time point at which arterial and portal venous enhancement is greatest. Time is shown in seconds. (Created with BioRender.com; M. Lee, 2025; <https://BioRender.com/inccxcl>.)



CT (Fig S3) (37,38). Diagnosis of AMI appears to be improved with DECT in the PVP, with improved sensitivity and reduced false-positives when using iodine maps and 40-keV monoenergetic images, which increase the conspicuity of ischemic segments (39), although data for clinical application are limited by the rarity of AMI and small sample sizes. More recently, iodine maps in the late arterial phase appear to significantly improve diagnostic accuracy for NOMI compared with that of conventional CT and PVP iodine maps (40). Additionally, DECT-derived iodine maps and virtual unenhanced images may increase sensitivity and reader confidence independent of experience and without an increase in interpretation time (41) (Fig S4).

Irrespective of technique, CT has the advantage of allowing assessment of alternative diagnoses in patients with suspected AMI. In a study of 959 individuals evaluated with CT angiography for suspected AMI, AMI was diagnosed in 19% of cases, whereas 61% had alternative diagnoses, including small-bowel obstruction (SBO) (10%), infectious colitis (9%), pneumonia (7%), cholecystitis (6%), and diverticulitis (6%) (42).

Interpretive Approach to AMI: How to Make the Diagnosis

A systematic interpretive approach to AMI based on underlying pathophysiology is imperative (Fig 5). Assessing the vasculature for the presence or absence of clot (ie, embolus or thrombosis) is the first crucial step to distinguish the type of AMI. If clot is identified in the SMA or SMV and there are no findings of strangulating bowel obstruction, the diagnosis of arterial or mesenteric venous occlusive AMI can be established. If there is no clot, the diagnosis of NOMI, occult clot, or small-vessel ischemia is possible. Next, assessment proceeds with bowel and extraintestinal findings to determine the distribution, extent, and severity of end-organ injury. Of course, the approach is flexible and bidirectional between the steps, since bowel and extraintestinal findings may be most conspicuous.

Inadequate Inflow: Arterial Occlusion

Arterial causes are the most common flow-related causes of AMI, accounting for 55%–70% of AMI cases (3,4). Arterial oc-

clusion comprises the majority of AMI cases and can be further subdivided into arterial embolism and thrombosis. AMI is differentiated from chronic mesenteric ischemia (CMI) by chronicity of symptom onset, cause of underlying vascular disease, and degree of blood flow compromise (11). CMI is almost invariably related to progressive atherosclerosis at the SMA origin and has a more indolent manifestation, characterized by postprandial abdominal pain, food aversion, and weight loss (26), and is not the focus of this article.

Arterial embolism is the most common cause of primary AMI overall, accounting for the majority of arterial occlusive AMI cases (1–3,11,17). Arterial emboli are typically located several centimeters beyond the SMA origin but can involve any part of the SMA (7), with classic pathognomonic sparing of the proximal SMA (9) (Figs 6, S5) and middle colic arterial branch. The SMA is susceptible due to its angulation from the aorta (27), and emboli typically lodge 3–15 cm from the SMA origin (9). Patients tend to be younger (6) and have underlying cardiac disease such as ischemic heart disease or atrial fibrillation. Less common causes include emboli from endocarditis, valvular disease, or aortic atheroma. Arterial embolic AMI tends to be more acute and painful, as the arterial collateral network has not developed compared with that in patients with underlying atherosclerosis and arterial thrombosis (14).

At CT angiography, arterial embolism appears as a low-attenuation filling defect in the SMA on contrast-enhanced images. The vessel cutoff sign refers to abrupt nonfilling of a vessel corresponding to the occlusive embolus (Figs 6, 7), and nonocclusive emboli may appear as eccentric filling defects. Given the typical sparing of the proximal SMA, the distal jejunum, ileum, and proximal colon are at risk for ischemia as opposed to the proximal jejunum, although bowel involvement depends on the location of occlusion. Importantly, 20%–68% of patients will have synchronous emboli involving other organs (43) (Fig 8). An accurate description of the precise location of arterial occlusion is imperative for treatment planning purposes in the setting of arterial embolism. In a recent retrospective review of original reports in 39 patients with arterial occlusive AMI, the location of occlusion was not mentioned in 62%, incorrect in 13%, incomplete in 8%, and appropriate in 18% (44).

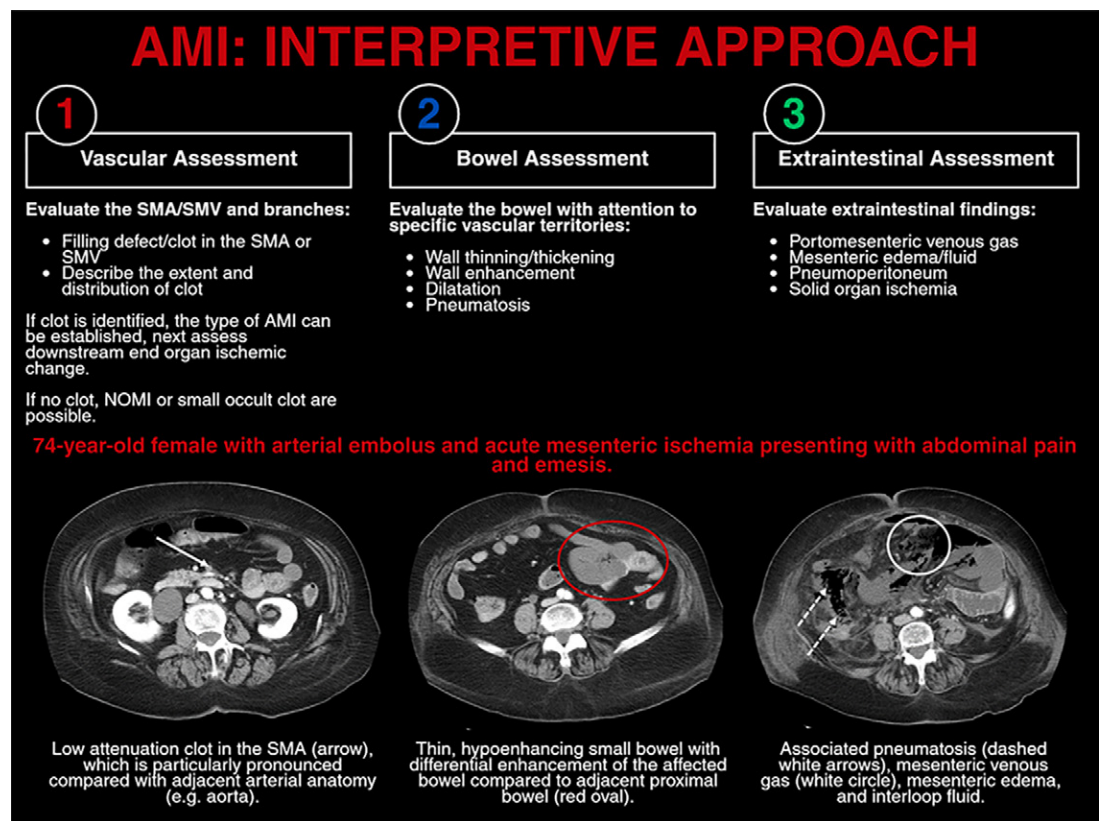


Figure 5. Interpretive approach to AMI. (Created with BioRender.com; M. Lee, 2025; <https://BioRender.com/hsjibr8>.)

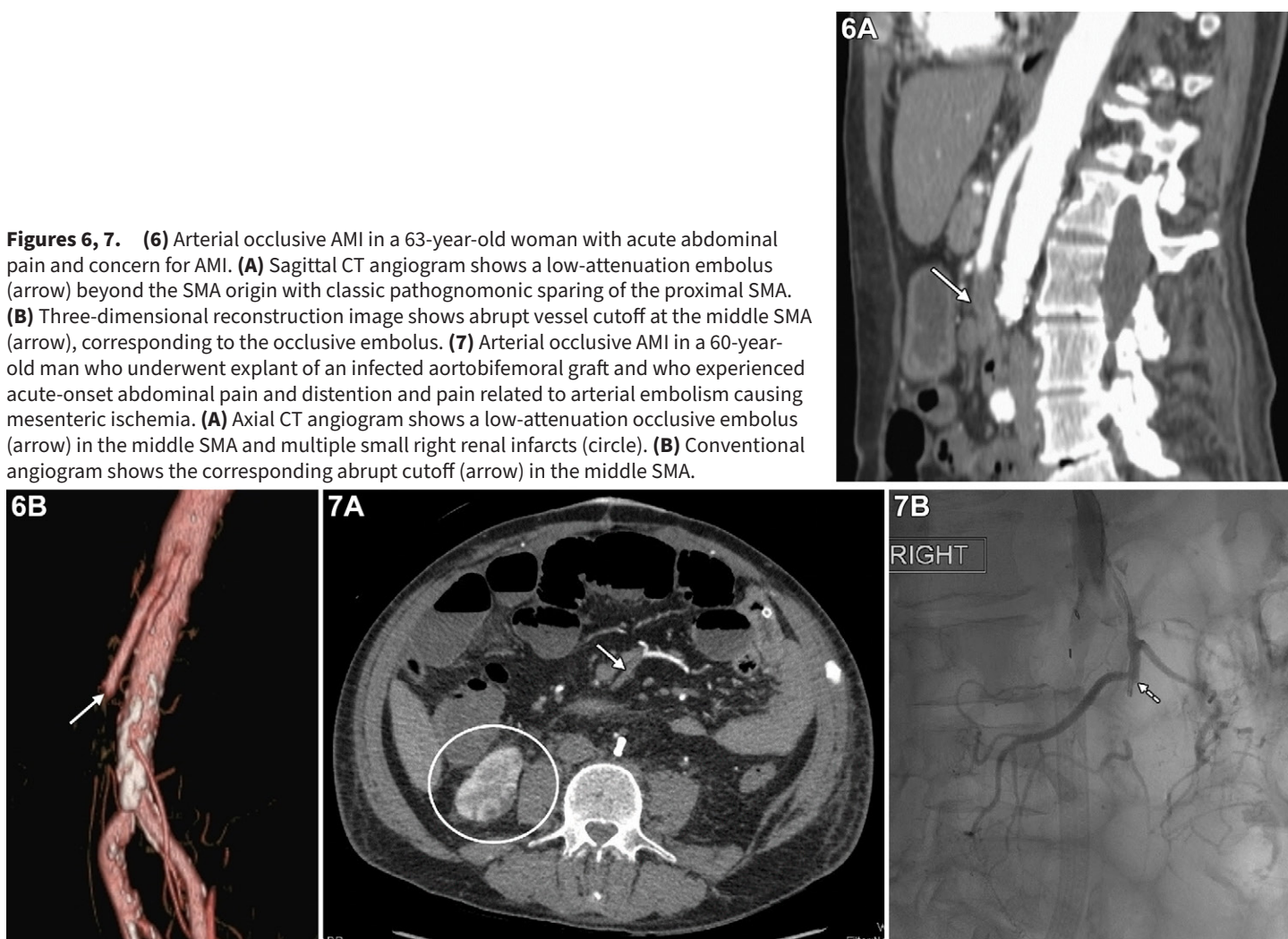
Arterial thrombosis is the other major cause of arterial occlusion, accounting for 15%–35% of AMI cases (1–3,9), with increased incidence in recent years (1). It tends to affect older patients, and underlying atherosclerosis is a major predisposing risk factor. Acute occlusion typically results from acute atherosclerotic plaque rupture (27). Given the chronicity of underlying atherosclerosis, patients often have collateralization. Other less common causes of arterial thrombosis include vasculitis (eg, polyarteritis nodosa) and segmental arterial mediolysis (45).

At CT angiography, in situ arterial thrombosis appears as a low-attenuation filling defect in the proximal SMA beginning at the origin (Figs 9, 10). There is often atherosclerosis (eg, calcification) and narrowing at the SMA origin (Fig 9). Given the proximal location of occlusion, there is typically more extensive bowel involvement. For example, involvement of the middle colic branch can lead to more distal colon ischemia (transverse colon to the splenic flexure) in addition to small-bowel ischemia. Because of the propensity for extensive bowel involvement, arterial thrombosis has the worst prognosis of AMI subtypes (12).

Decreased bowel wall enhancement is the hallmark feature of bowel ischemia in arterial occlusive AMI, with reported specificity of 97%–99% (46,47). Diminished or absent wall enhancement and thinning are specific to arterial causes of AMI (Figs 10, 11), not seen in venous causes. In early arterial occlusion, diminished wall enhancement is known as pale ischemia (11). Most of the time, ischemia is segmental and there are discrete transitions between ischemic and nonischemic bowel, which can be difficult to recognize unless one is aware and vigilant. Comparing suspected ischemic seg-

ments to adjacent normal loops can help to confidently identify decreased enhancement (Fig 12). Initially, ischemic loops of small bowel may be contracted in a phenomenon known as spastic reflex ileus (48). Extraintestinal findings such as mesenteric stranding are typically absent with arterial occlusive AMI as compared with NOMI and strangulated SBO, reported in as few as 10% of arterial occlusive AMI cases compared with 79%–93% for the other causes (49). Acutely, absence of mesenteric inflammation in early or nontransmural ischemia from arterial occlusion is an important pertinent negative that interpreting radiologists should be aware of in patients with possible AMI.

Arterial occlusion is itself a risk factor for and predictive of transmural necrosis (8,50,51). Although decreased or absent wall enhancement is characteristic of arterial AMI, it is not a statistically significant predictor of transmural necrosis (diagnostic odds ratio [DOR] = 7.9, $P = .12$). Diminished or absent enhancement in early arterial occlusive AMI is related to the underlying pathophysiology, but the wall may be viable and the injury still reversible (8). The severity of arterial vascular narrowing (ie, >70% stenosis, complete occlusion) and pneumatosis intestinalis are predictors of transmural necrosis (Fig 9) (51). Bowel dilatation and extreme bowel wall thinning with an imperceptible paper-thin appearance are predictive of transmural necrosis and related to irreversible injury to the deep muscle layers and myenteric plexus (Fig 12). Wall thinning is 38% sensitive and 98% specific for transmural necrosis (8). Bowel dilatation is more common in arterial occlusion than in venous occlusion or NOMI; importantly, bowel dilatation should be recognized as a predictor of transmural bowel necrosis in arterial occlusive AMI, not misinterpreted as simple ileus (8).



Figures 6, 7. (6) Arterial occlusive AMI in a 63-year-old woman with acute abdominal pain and concern for AMI. (A) Sagittal CT angiogram shows a low-attenuation embolus (arrow) beyond the SMA origin with classic pathognomonic sparing of the proximal SMA. (B) Three-dimensional reconstruction image shows abrupt vessel cutoff at the middle SMA (arrow), corresponding to the occlusive embolus. (7) Arterial occlusive AMI in a 60-year-old man who underwent explant of an infected aortobifemoral graft and who experienced acute-onset abdominal pain and distention and pain related to arterial embolism causing mesenteric ischemia. (A) Axial CT angiogram shows a low-attenuation occlusive embolus (arrow) in the middle SMA and multiple small right renal infarcts (circle). (B) Conventional angiogram shows the corresponding abrupt cutoff (arrow) in the middle SMA.

In a rare prospective cohort study, independent predictors of transmural necrosis requiring resection were organ failure (hazard ratio [HR] = 3.1, $P = .03$), serum lactate level greater than 2 mmol/L (HR = 4.1, $P = .01$), and bowel dilatation greater than 2.5 cm (HR = 2.6, $P = .02$) (50). In terms of posttreatment prognosis after first-line endovascular revascularization for arterial occlusive AMI, persistent bowel wall enhancement at initial CT and C-reactive protein (CRP) level less than 100 mg/L were independent predictors of 30-day intestinal resection-free survival (52). Additionally, bowel dilatation often precedes more well-known findings such as pneumatosis (53), and upstream gastric dilatation (gastric ileus) may be observed. Ultimately, interpreting radiologists should be vigilant for AMI as a potential cause of ileus at CT in a nonpost-surgical patient.

Inadequate Outflow: Mesenteric Venous Occlusion

Mesenteric venous occlusion, namely mesenteric venous thrombosis (MVT), accounts for 5%–20% of AMI cases (3,9,27,54). MVT typically occurs in younger patients who present with acute abdominal pain; acute MVT is symptomatic in about 90% of patients (7). MVT often results from a combination of hypercoagulability, endothelial injury, and

venous stasis related to local or systemic processes. Risk factors include underlying hypercoagulable states (54–56). The typical manifestation is different from that of arterial occlusive AMI; symptoms are more gradual and develop over days to weeks (9,27).

MVT—be it from inflammation and local factors contributing to large-vessel thrombosis or systemic hypercoagulability contributing to small-vessel thrombosis (56)—typically develops in smaller peripheral venous tributaries and propagates centrally (27), leading to impaired venous outflow with backup of blood and stasis. Although the bowel mucosa can initially augment oxygen extraction, increased hydrostatic pressure results in decreased arterial inflow, and intestinal capillaries are unable to provide adequate oxygen. Inflammation results in mucosal disruption and necrosis, allowing bacterial translocation, sepsis, hemodynamic instability, and multisystem organ failure (56).

MVT appears as a low-attenuation intraluminal filling defect in a mesenteric vein (Fig 13) with associated rim enhancement, attributable to the enhancing wall at contrast-enhanced CT (Fig 13) (12,54,57). Acute thrombus can be expansile high-attenuation thrombus on nonenhanced images. SMV thrombosis is more common (95%) than inferior mesenteric vein (IMV) thrombosis (4%–6%), and synchronous thrombosis

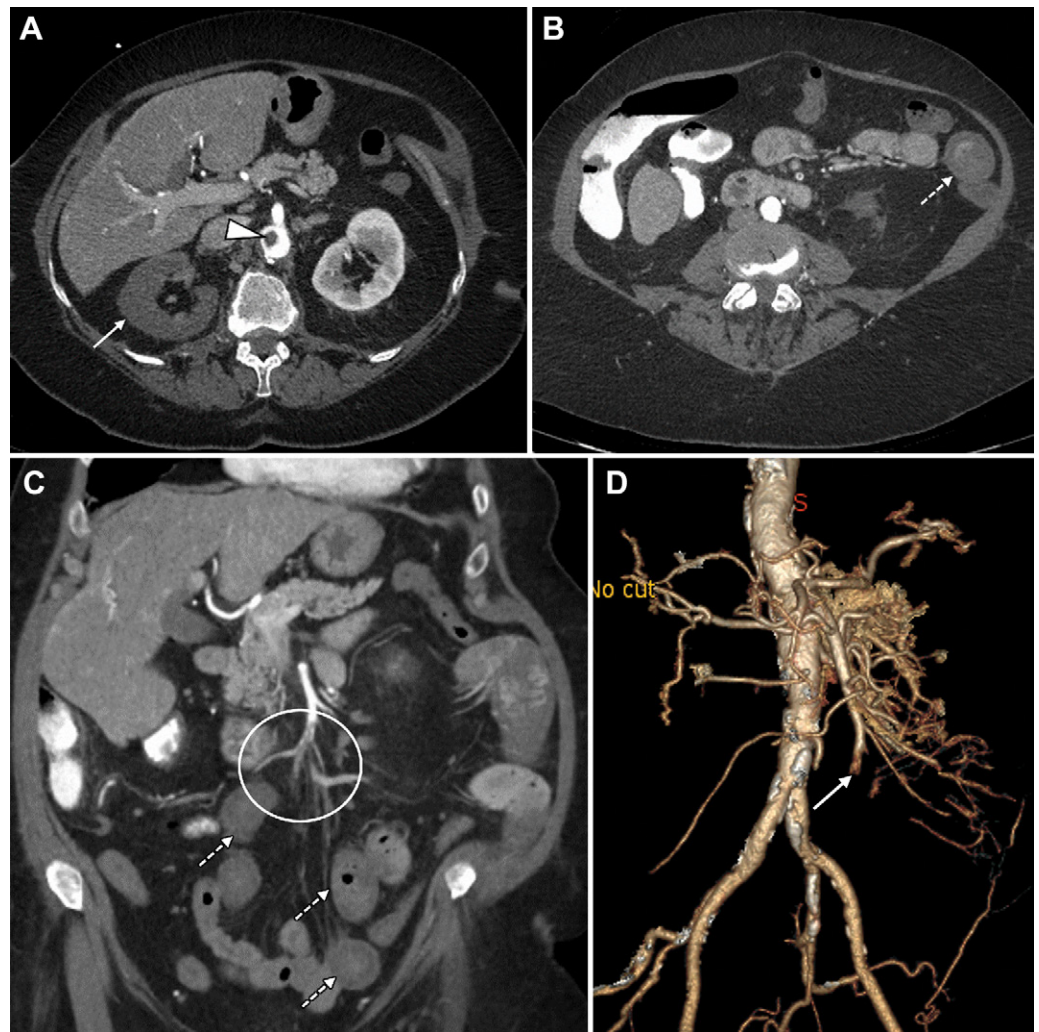


Figure 8. Arterial occlusive AMI in a 66-year-old woman with abdominal pain, acute mesenteric ischemia, and complete right renal infarct related to arterial emboli. **(A–C)** Axial **(A, B)** and coronal **(C)** CT angiograms show a nonenhancing right kidney indicative of infarction (arrow in **A**), mural thrombus in the aorta (arrowhead in **A**), extensive mesenteric arterial occlusion involving the middle SMA (circle in **C**), and corresponding hypo-enhancing loops of distal jejunum and ileum (arrows in **B** and **C**). There is preserved enhancement in the proximal jejunum. Associated wall thickening of the loops of hypo-enhancing bowel suggests an element of reperfusion injury. **(D)** Three-dimensional reconstruction image shows abrupt cutoff of the middle SMA (arrow). There is residual positive enteric contrast material in the colon from a prior study.

of the splenic and portal veins is common. The ileum is most commonly involved (64%–83% of cases), followed by the jejunum (50%–81%) and duodenum (4%–8%) (56). More peripheral thrombosis involving smaller mesenteric vein branches has greater risk for developing transmural necrosis, related to fewer collaterals (27,57).

Circumferential bowel wall thickening related to intramural edema and hemorrhage is a characteristic imaging feature of mesenteric venous ischemia, and wall thickening can be marked, up to 1.5 cm (normal, 0.3–0.5 cm) (54,57). Wall edema from venous congestion and increased intramural hydrostatic pressure from continued arterial inflow and obstructed venous outflow results in mural stratification and the target or halo cross-sectional appearance of the bowel: alternating high, low, and high attenuation corresponding to the mucosa, submucosal edema, and muscularis propria, respectively (Figs 13, S6) (57). Mural stratification is more common in mesenteric venous AMI than in arterial AMI (57). If seen in arterial AMI, it is due to reperfusion injury with restoration of arterial inflow. Extraintestinal findings are also pronounced in MVT, with mesenteric haziness, edema, and fluid being common and typically much more conspicuous than in arterial causes (Fig S6) (57).

Decreased or absent bowel wall enhancement (diagnostic odds ratio [DOR] = 8.2, $P = .00$) and bowel dilatation (DOR =

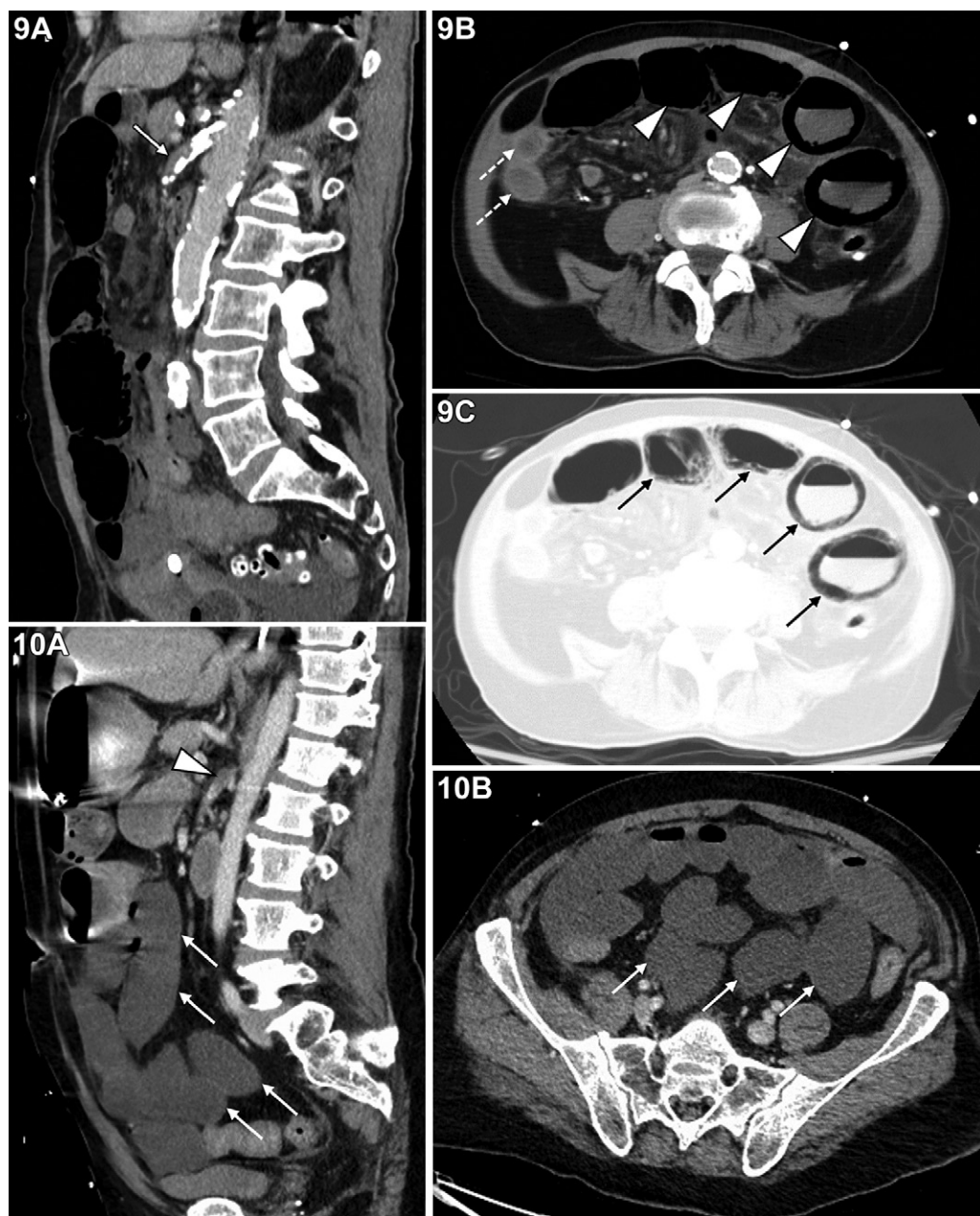
3.1, $P = .00$) are statistically significant predictors of transmural necrosis in venous AMI (8,58–60). Decreased or absent wall enhancement reflects cessation of arterial flow related to later-stage ischemia. Importantly, the degree of wall thickening and extraintestinal findings do not correlate with the severity of ischemic bowel injury (14).

Global Hypoperfusion: NOMI

NOMI is the main cause of AMI in critically ill patients and accounts for 20%–30% of AMI cases (11), although the true prevalence is likely higher because it is difficult to diagnose compared with other forms of AMI and is likely underrecognized (4). A recent multicenter observational study that assessed the incidence, management, and outcomes of AMI found that nearly 60% of sites did not include any patients with NOMI (4). NOMI encompasses a multifactorial process whereby global hypoperfusion (eg, low-flow states, hypotension) results in AMI (61). As the name implies, NOMI occurs in the absence of vascular occlusion.

Predisposing risk factors for NOMI include critical illness and shock (eg, hemorrhagic, cardiogenic, septic); vasoactive medications (eg, epinephrine, norepinephrine, vasopressin), which increase systemic blood pressure but fail to increase mesenteric blood flow; and other vasoactive drugs

Figures 9, 10. (9) SMA thrombosis and AMI in a 77-year-old man. Sagittal (A) and axial (B, C) contrast-enhanced CT images in soft-tissue window (B) and lung window (C) show proximal arterial thrombosis and occlusion at the SMA origin in the setting of severe atherosclerosis (arrow in A). There is extensive transmural ischemia involving loops of small bowel throughout the central and left abdomen, with extensive small-bowel pneumatosis (arrows in C) and absent wall enhancement (arrowheads in B). There are normally enhancing loops of bowel (arrows in B) in the right abdomen. (10) SMA thrombosis and AMI in a 49-year-old man. Sagittal (A) and axial (B) contrast-enhanced CT images show proximal arterial occlusion at the SMA origin (arrowhead in A) with extensive thin-walled, nonenhancing, dilated and fluid-filled loops of bowel (arrows). Bowel dilatation should be recognized as a predictor of transmural bowel necrosis in the setting of arterial occlusive AMI, not misinterpreted as simple ileus.



(eg, cocaine) (9). Hemodialysis in patients with end-stage renal disease is an additional risk factor for NOMI from hypotension during hemodialysis (62). A characteristic feature of NOMI is multisystem involvement (eg, bowel, liver, renal injury) compared with early arterial occlusive AMI, which is typically limited to bowel injury (Fig 14) (61). NOMI is associated with high mortality (30%–93%) and poor prognosis (11), in part related to delay in diagnosis and development of transmural necrosis and multiorgan failure (61), in addition to coexisting comorbidities and critical illness.

Characteristic imaging features of NOMI are attenuated, irregular-appearing, but patent vessels with areas of narrowing (Fig 15). CT angiographic findings mirror angiographic findings of mesenteric vasoconstriction, including narrowing of SMA branches with alternate dilatation and narrowing and a resultant “string of sausages” sign; mesenteric arterial spasm; and impaired vascular filling (14). Typically, there is a

widespread discontinuous pattern of bowel ischemia and necrosis (14). The discontinuous or segmental pattern typically involves multiple vascular territories with coexisting ischemia, reperfusion, and necrosis (12). Comparison with prior imaging studies (ie, before critical illness) can be helpful to identify a change in mean diameter of the SMA (63). Given the underlying pathophysiology, findings of CT hypoperfusion complex and “shock bowel” may be present (11). A 50% reduction in blood flow through the SMA initially results in vasodilatation; but with persistent hypoperfusion, mesenteric vasoconstriction exacerbates ischemia (63–65). The pattern of bowel enhancement can be variable, and mesenteric stranding and ascites are common (7).

Imaging features of transmural necrosis in NOMI are often grouped with arterial occlusive AMI as “arterial origin AMI” (8); however, the clinical and pathophysiologic characteristics and patterns of bowel ischemia are different (53).

Figure 11. Arterial occlusive AMI in a 55-year-old woman with a history of renal transplant and arterial thrombosis. Sagittal (A) and axial (B) contrast-enhanced CT images show a low-attenuation SMA thrombus (solid arrow) and background calcified atherosclerosis. There is associated nonenhancement of small-bowel jejunal loops in the left abdomen (circle in B). There is a sharp transition between enhancing or perfusing loops (dashed arrow) and nonenhancing loops. There is faint peritoneal enhancement (arrowhead in B) adjacent to the nonenhancing bowel.

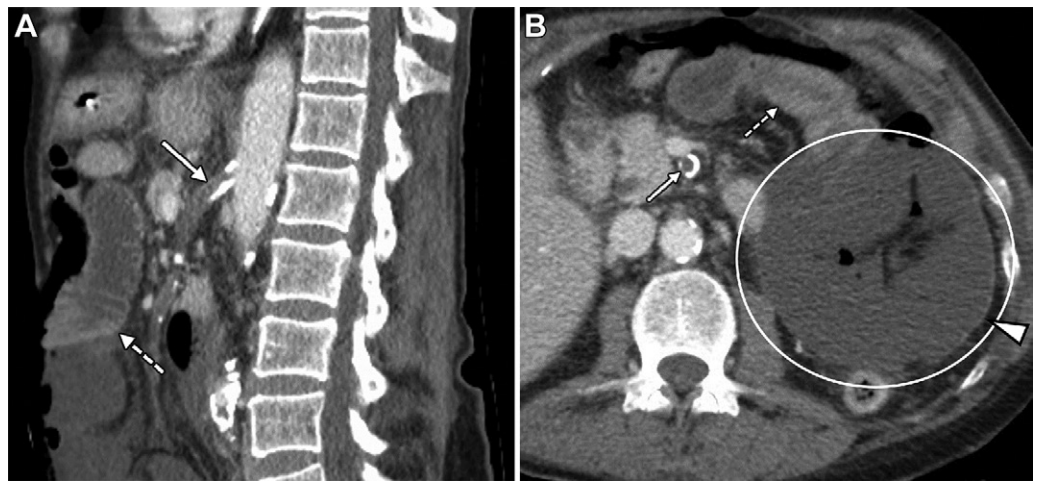
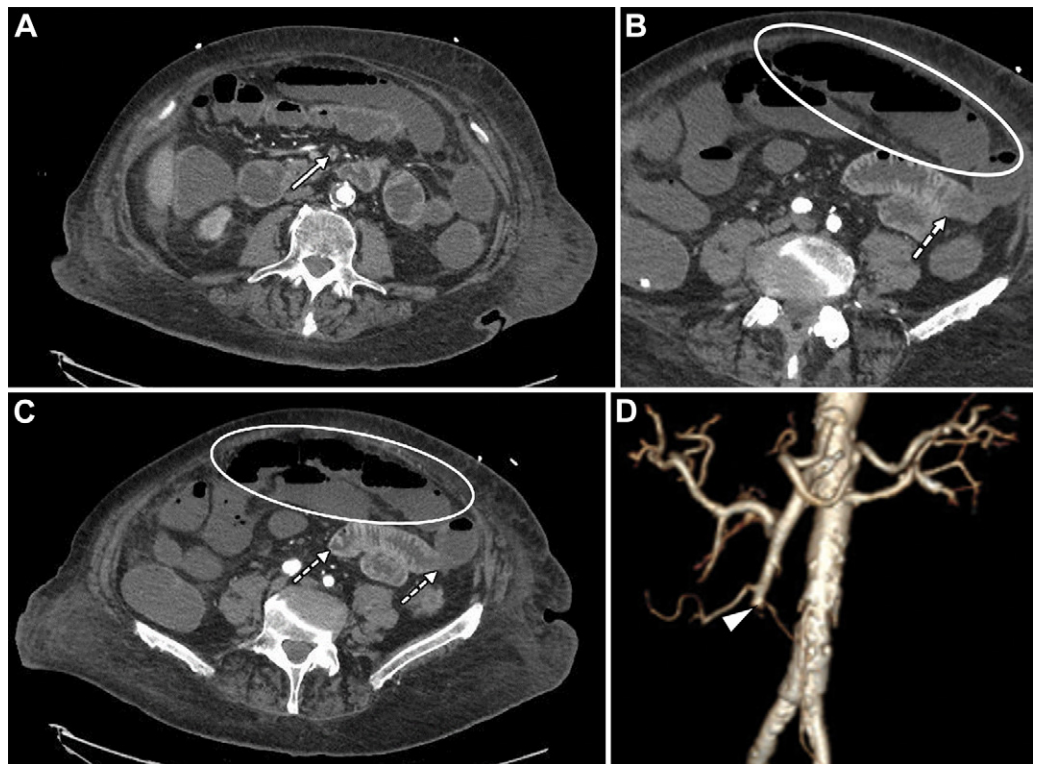


Figure 12. Characteristic imaging features of AMI from arterial occlusion in an 83-year-old man with a history of atrial fibrillation and arterial embolus. (A–C) Axial CT angiograms show a clot filling defect in the middle SMA (arrow in A), with corresponding nonenhancing or hypoenhancing dilated and fluid-filled loops of distal jejunum and ileum with paper-thin imperceptible walls (ovals in B and C). There is a sharp transition (arrows in B and C) between enhancing or perfusing loops and non-enhancing or hypoenhancing ischemic loops. The paper-thin imperceptible wall is predictive of transmural necrosis. (D) Three-dimensional reconstruction image shows abrupt cutoff of the middle SMA (arrowhead).



Absence of bowel enhancement is the most consistent feature of transmural necrosis in NOMI (66). Additionally, although NOMI can affect the entire distribution of the gastrointestinal tract, a more proximal distribution of ischemia in the gastrointestinal tract is associated with worse prognosis (66), presumably related to more severe hemodynamic impairment, since the proximal gastrointestinal tract (eg, the stomach) has robust collateralization and is typically less susceptible to ischemia. In contrast to arterial occlusive AMI, in NOMI bowel dilatation is confounded in a critically ill patient who may have coexisting ileus or other reasons to have bowel dilatation (53).

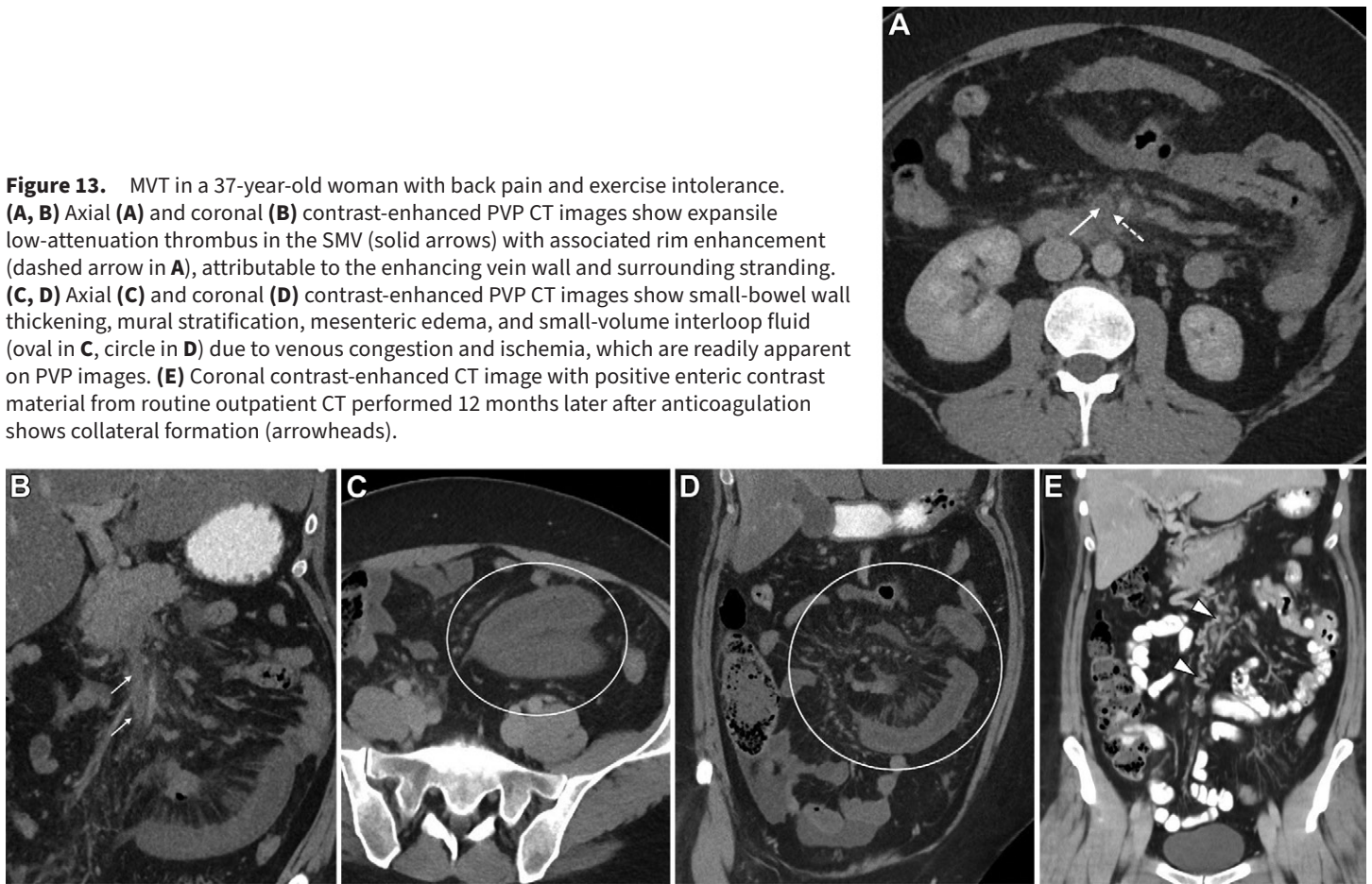
In a retrospective study of 154 patients with NOMI, CT features associated with transmural necrosis were absent wall enhancement ($P < .001$), wall thinning ($P < .001$), bowel dilatation ($P = .041$), pneumatosis ($P = .003$), mesenteric gas ($P =$

.005), and portal venous gas ($P = .002$) (61). Using clinical and imaging findings associated with irreversible transmural necrosis, Calame et al (61) identified four predictive factors—absent bowel enhancement, bowel thinning, plasma bicarbonate concentration of 15 mmol/L or less, and prothrombin rate less than 40%—to devise a system for risk stratification whereby 88% and 100% of patients with three or four of these features, respectively, had irreversible transmural bowel necrosis (61). In patients with NOMI undergoing cardiopulmonary bypass, pneumatosis, bowel loop dilatation, and differential mural enhancement were associated with a 98% probability of ischemic bowel (67).

Strangulating Bowel Obstruction

SBO is complicated by bowel ischemia in ~10% of cases (25) and is commonly related to closed-loop obstruction (CLO)

Figure 13. MVT in a 37-year-old woman with back pain and exercise intolerance. (A, B) Axial (A) and coronal (B) contrast-enhanced PVP CT images show expansile low-attenuation thrombus in the SMV (solid arrows) with associated rim enhancement (dashed arrow in A), attributable to the enhancing vein wall and surrounding stranding. (C, D) Axial (C) and coronal (D) contrast-enhanced PVP CT images show small-bowel wall thickening, mural stratification, mesenteric edema, and small-volume interloop fluid (oval in C, circle in D) due to venous congestion and ischemia, which are readily apparent on PVP images. (E) Coronal contrast-enhanced CT image with positive enteric contrast material from routine outpatient CT performed 12 months later after anticoagulation shows collateral formation (arrowheads).



(49). *Strangulated obstruction* is defined as obstruction with blood flow compromise and ischemia (25), and CLO occurs when a loop of bowel is obstructed at two points, resulting in a sequestered loop of bowel, which is at high risk for ischemia (68). Mortality for patients undergoing surgery for SBO with ischemia is as high as 25% (68), and CLO is the most common cause of bowel ischemia overall. Adhesions and hernias are the most common causes of CLO (25,49). Bowel ischemia in CLO is multifactorial, related to compression of the vascular pedicle, distention of the closed loop, increased transmural hydrostatic pressure and wall tension, and ultimately decreased blood flow within the local venous vessels, then arterial vessels (69).

Characteristic imaging features of CLO include the “double beak” sign, a C- or U-shaped configuration of obstructed bowel, a radial configuration of dilated bowel with a “balloons on a string” appearance (Fig 16), and the whirl sign when there is volvulus (68). In a meta-analysis of CT findings of strangulating SBO, reduced bowel wall enhancement was the most specific finding, with specificity of 95% and positive likelihood ratio (LR) of 11. The “mesenteric fluid” sign—defined as hazy fluid attenuation in the mesentery of the involved intestinal segment, resulting from venous congestion and mesenteric venous outflow obstruction—had a sensitivity of 89% with a negative LR of 0.16 (70).

In a retrospective series of 256 patients with adhesive SBO, three CT findings were significantly associated with strangulation: decreased bowel wall enhancement relative to unin-

involved bowel (OR = 7.8; 95% CI: 2.6, 23.5), diffuse mesenteric haziness (OR = 6.1; 95% CI: 2.5, 15.2), and CLO (OR = 6.5; 95% CI: 2.8, 15.5) (Fig S7). The combination of these three findings had an area under the curve (AUC) of 0.91 (95% CI: 0.86, 0.96) and negative predictive value of 97% (95% CI: 93%, 99%) (25). The combination of two or all three findings strongly suggests surgical intervention, whereas absence of these findings effectively rules out strangulation and suggests that the SBO can be managed without surgery (25).

In a recent retrospective study of 145 patients with CLO from adhesion or internal hernia, increased attenuation of intestinal contents (>8 HU) and the intestinal wall (>5.8 HU) (ie, difference between the involved loop and proximal dilated loop), diffuse mesenteric haziness, and mesenteric fluid were independent predictors of irreversible bowel necrosis, with OR = 3.9–45.3 (*P* value range, <.001–.045) (69). Increased attenuation of intraluminal bowel contents was 64% sensitive and 99% specific for bowel necrosis (Fig 17), and increased attenuation of the bowel wall was 67% sensitive and 92% specific for bowel necrosis (69). High attenuation presumably represents extravasation of blood into the wall and lumen from progressive ischemia, wall breakdown, and permeability.

Points of Clarification: Reperfusion Injury and Pneumatosis Intestinalis

Bowel wall thickening is a frequently reported sensitive but nonspecific response of bowel to ischemia (10,71), but it

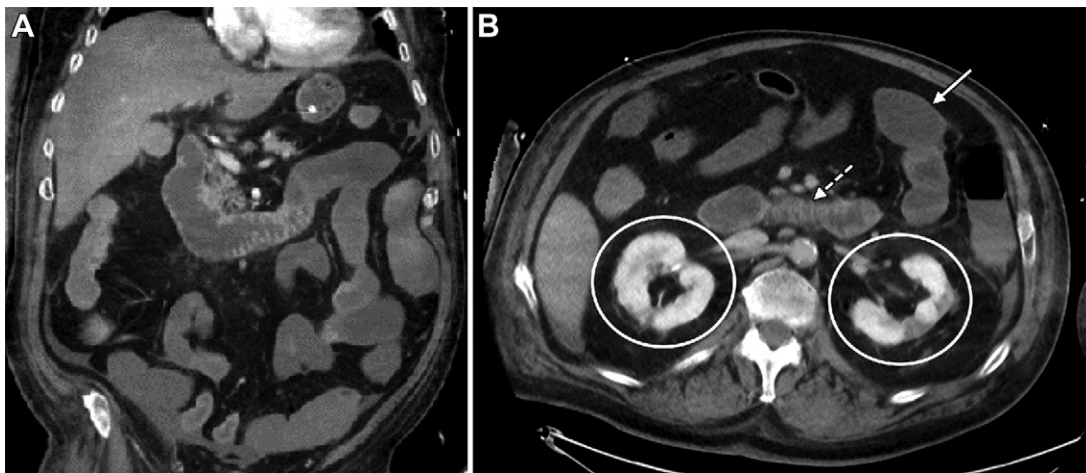
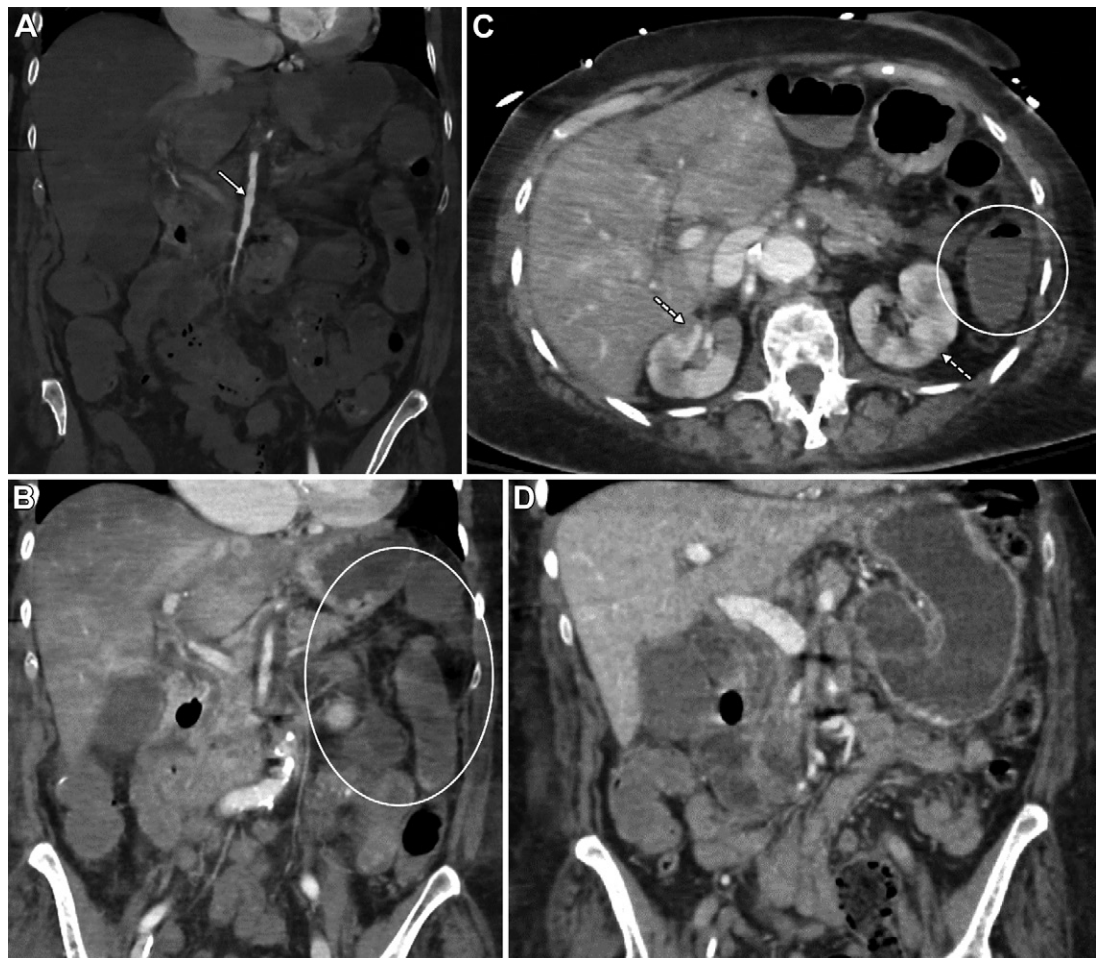


Figure 14. Hemorrhagic shock causing NOMI in a 79-year-old critically ill man. Coronal (**A**) and axial (**B**) contrast-enhanced CT images show a widespread discontinuous pattern of bowel ischemia, with segmental hypo- and nonenhancing bowel (solid arrow in **B**) affecting multiple vascular territories (small bowel and colon). The affected bowel is thin walled and fluid filled. Differential enhancement is most notable compared with the enhancing duodenum (dashed arrow in **B**). Multifocal renal infarcts (circles in **B**) are indicative of multisystem involvement. There is also renal hyperenhancement related to shock. The patient died 1 day later.

Figure 15. Hemorrhagic shock causing NOMI in an 83-year-old woman. (**A–C**) Coronal CT angiogram (**A**) and coronal (**B**) and axial (**C**) contrast-enhanced CT images show a patent but attenuated, irregular-appearing SMA (arrow in **A**). There is widespread bowel ischemia with segmental areas of hypo- and nonenhancing bowel affecting multiple vascular territories. The ischemic bowel is thin walled and fluid filled (oval in **B**, circle in **C**) and hypoenhancing compared with small bowel in the right abdomen. Multifocal renal infarcts (arrows in **C**) are indicative of multisystem involvement. (**D**) Coronal contrast-enhanced CT image from 3 days earlier shows nondilated normally enhancing loops of bowel. Comparison with imaging studies obtained before critical illness can be helpful for assessing interval change and findings of transmural necrosis. The patient died 3 days later.



depends on the underlying cause of AMI and the time course of injury (65). For example, wall thickening in arterial occlusive AMI more likely represents reperfusion injury, which can be seen in up to 44% of patients after endovascular revascular-

ization (72). More than 90% of patients with reperfusion injury have bowel wall thickening (~0.9 cm), submucosal edema, mucosal hyperenhancement, and mesenteric stranding (72) (Fig 18). Decreased bowel wall enhancement at pretreatment

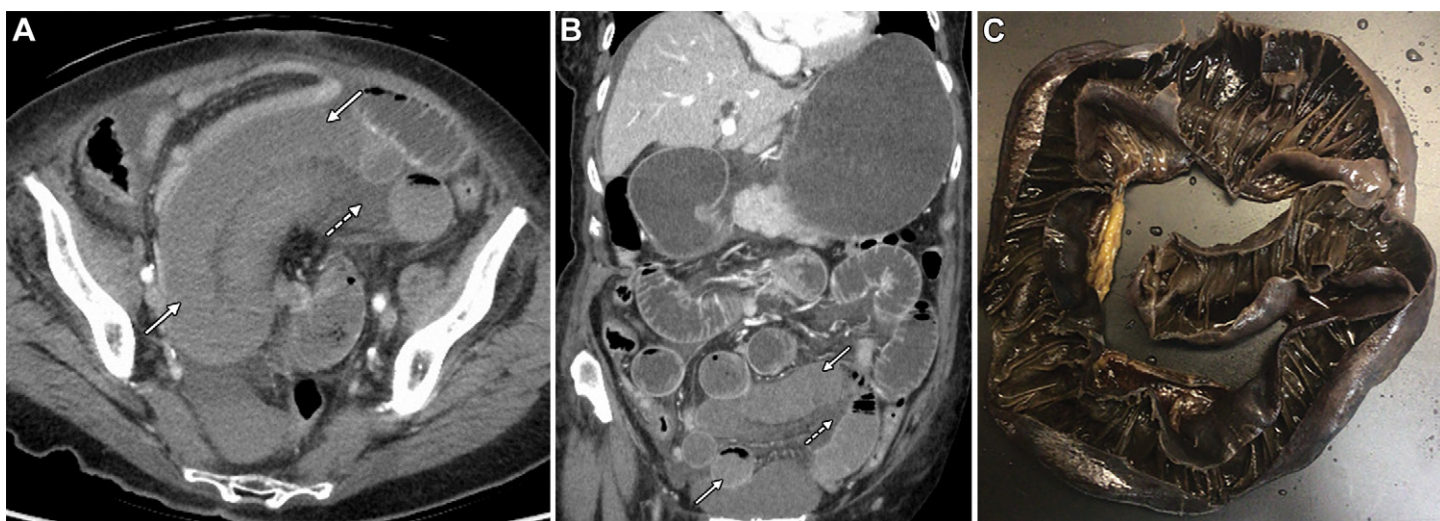


Figure 16. CLO in a 77-year-old man. (A, B) Axial (A) and coronal (B) contrast-enhanced CT images show an obstructed, U-shaped, fluid-filled, sequestered loop of bowel in the pelvis with high-attenuation intraluminal contents (solid arrows), compared with adjacent proximal loops of bowel and associated mesenteric haziness and edema (dashed arrow). Intraoperatively, the closed-loop segment was hemorrhagic appearing and torsed around a single adhesive band in the pelvis. (C) Gross specimen photograph shows approximately 75 cm of resected infarcted bowel.

CT (hazard ratio [HR] = 8.2; 95% CI: 2.0, 33.0) (72), arterial embolism (HR = 7.4; 95% CI: 1.5, 34.7), and complete SMA occlusion (HR = 7.0; 95% CI: 1.0, 45.5) were independent predictors of reperfusion injury in arterial occlusive AMI (72). Reperfusion injury can mimic new or ongoing AMI but does not appear to affect short-term survival, and awareness of this entity may avoid unnecessary surgery and bowel resection.

Pneumatosis, portomesenteric venous gas, and pneumoperitoneum are classically indicative of irreversible transmural necrosis (73), as mural necrosis allows gas to pass from the mucosa to the submucosa and into the mesenteric veins (Fig 19). Of these, pneumatosis is often the most perplexing because of its association with life-threatening ischemia as well as a spectrum of “benign” causes. This is in part because CT enables detection of subtle pneumatosis at earlier stages of disease, and isolated pneumatosis can be seen with partial mural ischemia (ie, viable bowel) without transmural necrosis (73). As such, the presence of pneumatosis cannot be interpreted in isolation but should be considered in context and supported by additional imaging, clinical, and laboratory findings when assessing for transmural necrosis.

In a recent study of patients with NOMI undergoing surgery for suspected transmural necrosis, among patients with pneumatosis, no patients with normal wall enhancement had transmural necrosis, whereas 77% of patients with absent wall enhancement did (61). Bandlike pneumatosis and the combination of pneumatosis and portal venous gas are highly associated with transmural infarct (Fig 9) (74). Features predicting need for surgery in patients with pneumatosis include abdominal distention or dilated loops of bowel at CT (OR = 13.2, $P = .001$), peritonitis (OR = 9.4, $P = .007$), and lactic acidemia (OR = 2.3, $P = .02$) (75).

Conclusion

AMI is a rare abdominal emergency that has high morbidity and mortality. Radiologists who interpret abdominal CT studies are critical for assessment of patients with AMI and should

be comfortable making this important diagnosis. The role of radiology cannot be overstated, as clinical features are often nonspecific and there are no reliable laboratory tests or biomarkers for early diagnosis, to our knowledge.

The authors of this article have shown how to contextualize imaging findings of AMI through a pathophysiology- rather than semiology-based approach emphasizing the imaging features predictive of transmural necrosis, which are different based on the underlying cause of AMI and are important for establishing a critical diagnosis and informing prognosis. The sentiment that AMI encompasses processes for which the “diagnosis is impossible, the prognosis hopeless, and the treatment useless”—expressed by Cokkinis (76) nearly a hundred years ago—reverberates today, with undoubted room for improvement, although the authors hope that this work will empower interpreting radiologists with interpretive skills to make an important diagnosis and point toward a more hopeful future.

Author affiliations:

¹ Department of Radiology, University of Wisconsin School of Medicine and Public Health, 600 Highland Ave, Madison, WI 53792

² Department of Pathology and Laboratory Medicine, University of Wisconsin School of Medicine and Public Health, Madison, Wis

Received February 1, 2025; revision requested March 17 and received April 21; accepted April 25. Presented as an education exhibit at the 2024 RSNA Annual Meeting.

Address correspondence to: M.H.L. (email: mlee5@uwhealth.org).

Disclosures of conflicts of interest.—**M.H.L.** Spouse is stockholder in Exact Sciences. **P.J.P.** Consultant for Bracco Diagnostics, GE HealthCare, and Nanox-AI; stockholder in ColoWatch. **G.V.T.** Consultant for GE HealthCare and Canon Medical, payment or honoraria from GE HealthCare and Canon Medical. **M.G.L.** Spouse is a consultant for Elephas Biosciences; 2023–2024 Igor Laufer visiting professor, supported by Society of Abdominal Radiology and Bracco. **D.H.K.** Board member of Society of Abdominal Radiology, stockholder in Elucant LLC. All other authors, the editor, and the reviewers have disclosed no relevant relationships.

References

1. Bala M, Catena F, Kashuk J, et al. Acute mesenteric ischemia: updated guidelines of the World Society of Emergency Surgery. *World J Emerg Surg* 2022;17(1):54.

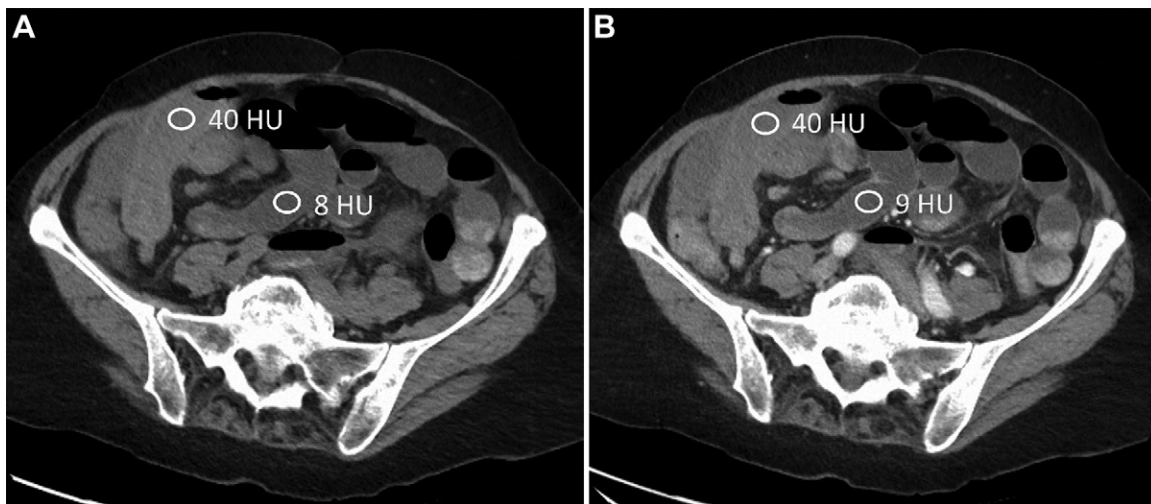
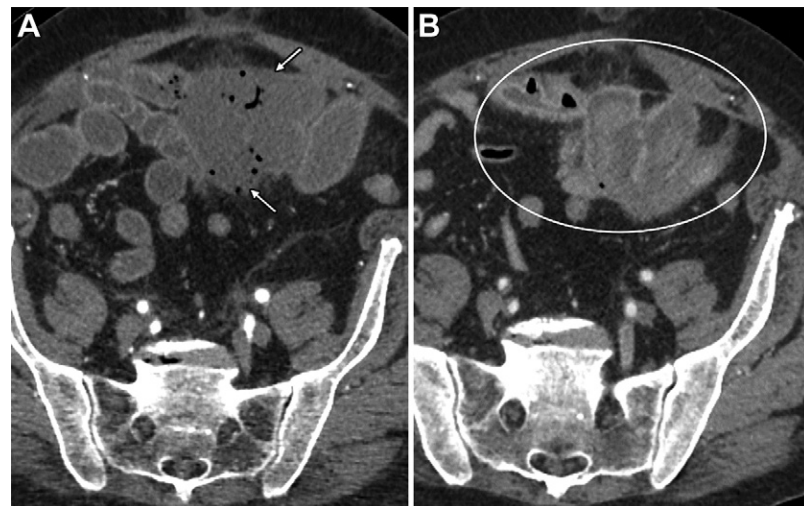


Figure 17. Closed-loop SBO in an 85-year-old woman with pelvic pain secondary to the SBO. Axial noncontrast (**A**) and contrast-enhanced (**B**) CT images of the abdomen show dilated fluid-filled loops of bowel in the pelvis. High-attenuation contents within the obstructed sequestered loop of bowel probably represent necrotic bowel with intraluminal hemorrhage. Lower-attenuation fluid contents are noted in the upstream loops of small bowel in the left pelvis. The attenuation of the intraluminal contrast material was unchanged between noncontrast and contrast-enhanced imaging. The patient was taken emergently to the operating room, where CLO and small-bowel necrosis were confirmed, with subsequent small-bowel resection.

Figure 18. SMA embolus in a 68-year-old man with esophageal cancer. (**A**) Axial contrast-enhanced CT angiogram shows thin nonenhancing loops of bowel (arrows) in the pelvis. (**B**) Follow-up axial contrast-enhanced CT image 3 weeks later shows wall thickening of the ischemic loops (oval), related to reperfusion injury.



- Clair DG, Beach JM. Mesenteric Ischemia. *N Engl J Med* 2016;374(10):959–968.
- Tamme K, Reintam Blaser A, Laisaar KT, et al. Incidence and outcomes of acute mesenteric ischaemia: a systematic review and meta-analysis. *BMJ Open* 2022;12(10):e062846.
- Reintam Blaser A, Mändul M, Björck M, et al; AMESI Investigators (Collaborators). Incidence, diagnosis, management and outcome of acute mesenteric ischaemia: a prospective, multicentre observational study (AMESI Study). *Crit Care* 2024;28(1):32.
- Molyneux K, Beck-Esmay J, Koyfman A, Long B. High risk and low prevalence diseases: mesenteric ischemia. *Am J Emerg Med* 2023;65:154–161.
- Schoots IG, Koffeman GI, Legemate DA, Levi M, van Gulik TM. Systematic review of survival after acute mesenteric ischaemia according to disease aetiology. *Br J Surg* 2004;91(1):17–27.
- Garzelli L, Ben Abdallah I, Nuzzo A, et al. Insights into acute mesenteric ischaemia: an up-to-date, evidence-based review from a mesenteric stroke centre unit. *Br J Radiol* 2023;96(1151):20230232.
- Zeng Y, Yang F, Hu X, Zhu F, Chen W, Lin W. Radiological predictive factors of transmural intestinal necrosis in acute mesenteric ischemia: systematic review and meta-analysis. *Eur Radiol* 2023;33(4):2792–2799.
- Theodore S, Xia T, Saillant N. Intestinal Ischemia: Etiology and Foundational Concepts. *NEJM Evid* 2024;3(3):EVIDra2300266.
- Furukawa A, Kanasaki S, Kono N, et al. CT Diagnosis of Acute Mesenteric Ischemia from Various Causes. *AJR Am J Roentgenol* 2009;192(2):408–416.
- Dhatt HS, Behr SC, Miracle A, Wang ZJ, Yeh BM. Radiological Evaluation of Bowel Ischemia. *Radiol Clin North Am* 2015;53(6):1241–1254.
- Fitzpatrick LA, Rivers-Bowerman MD, Thippavong S, Clarke SE, Rowe JA, Costa AF. Pearls, Pitfalls, and Conditions That Mimic Mesenteric Ischemia at CT. *RadioGraphics* 2020;40(2):545–561.
- Gore RM, Yaghamai V, Thakrar KH, et al. Imaging in Intestinal Ischemic Disorders. *Radiol Clin North Am* 2008;46(5):845–875, v.
- Kanasaki S, Furukawa A, Fumoto K, et al. Acute Mesenteric Ischemia: Multidetector CT Findings and Endovascular Management. *RadioGraphics* 2018;38(3):945–961.
- Mallick IH, Yang W, Winslet MC, Seifalian AM. Ischemia-reperfusion injury of the intestine and protective strategies against injury. *Dig Dis Sci* 2004;49(9):1359–1377.
- Brandt LJ, Feuerstadt P, Longstreth GF, Boley SJ; American College of Gastroenterology. ACG Clinical Guideline: Epidemiology, Risk Factors, Patterns of Presentation, Diagnosis, and Management of Colon Ischemia (CI). *Am J Gastroenterol* 2015;110(1):18–44; quiz 45.
- Sinha D, Kale S, Kundaragi NG, Sharma S. Mesenteric ischemia: a radiologic perspective. *Abdom Radiol (NY)* 2022;47(5):1514–1528.

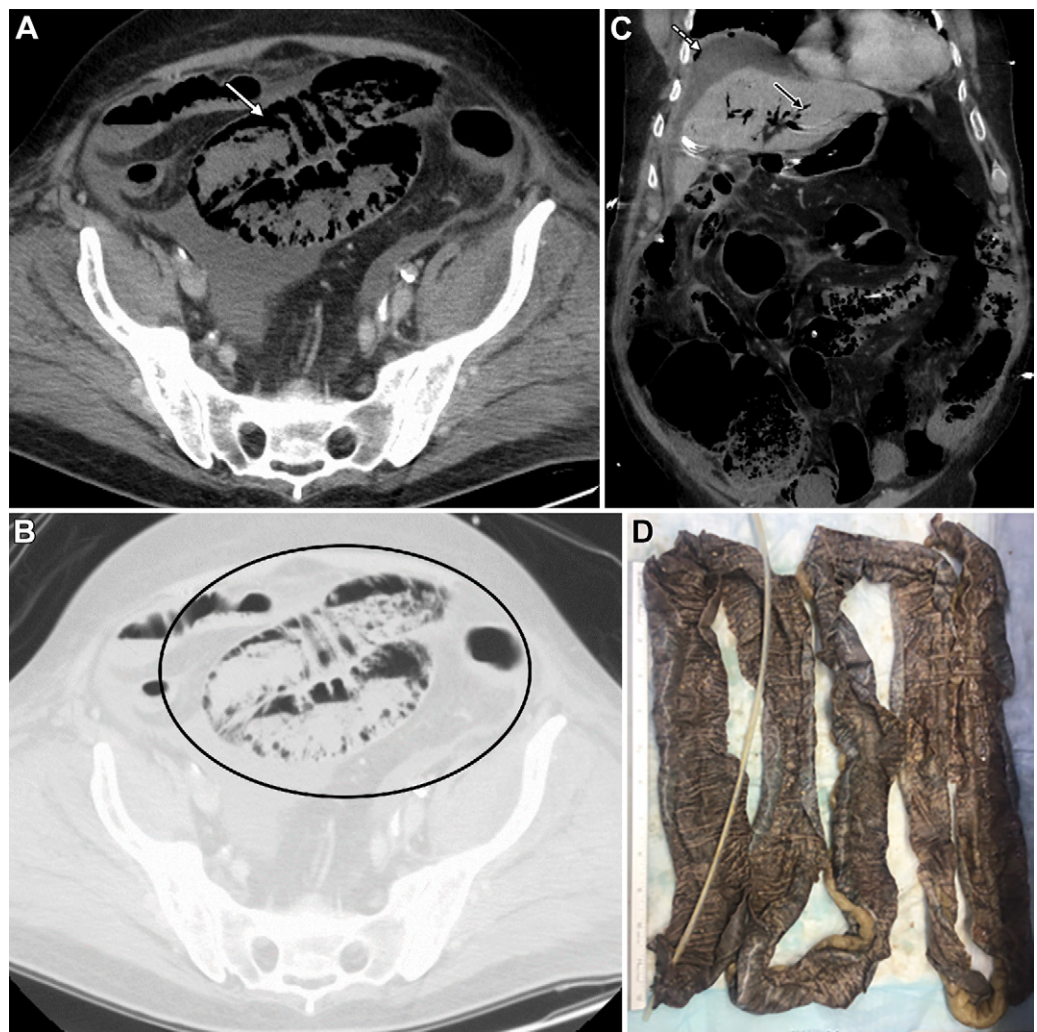


Figure 19. AMI in a 72-year-old man with atrial fibrillation. (A–C) Axial soft-tissue window (A), axial lung window (B), and coronal (C) contrast-enhanced CT images show extensive pneumatosis (arrow in A, oval in B), portal venous gas (black arrow in C), free fluid, and foci of pneumoperitoneum (white arrow in C). (D) Gross specimen photograph shows 260 cm of necrotic bowel.

18. Theodore S, Xia T, Saillant N. The Evaluation and Management of Intestinal Ischemia. *NEJM Evid* 2024;3(4):EVIDra2400057.
19. Acosta S, Nilsson T. Current status on plasma biomarkers for acute mesenteric ischemia. *J Thromb Thrombolysis* 2012;33(4):355–361.
20. Nuzzo A, Joly F, Ronot M, et al; SURVI group. Normal Lactate and Unenhanced CT-Scan Result in Delayed Diagnosis of Acute Mesenteric Ischemia. *Am J Gastroenterol* 2020;115(11):1902–1905.
21. Montagnana M, Danese E, Lippi G. Biochemical markers of acute intestinal ischemia: possibilities and limitations. *Ann Transl Med* 2018;6(17):341.
22. Tilsed JVT, Casamassima A, Kurihara H, et al. ESTES guidelines: acute mesenteric ischaemia. *Eur J Trauma Emerg Surg* 2016;42(2):253–270.
23. Aliosmanoglu I, Gul M, Kapan M, et al. Risk factors effecting mortality in acute mesenteric ischemia and mortality rates: a single center experience. *Int Surg* 2013;98(1):76–81.
24. Adaba F, Askari A, Dastur J, et al. Mortality after acute primary mesenteric infarction: a systematic review and meta-analysis of observational studies. *Colorectal Dis* 2015;17(7):566–577.
25. Millet I, Boutot D, Faget C, et al. Assessment of Strangulation in Adhesive Small Bowel Obstruction on the Basis of Combined CT Findings: Implications for Clinical Care. *Radiology* 2017;285(3):798–808.
26. Lam A, Kim YJ, Fidelman N, et al; Expert Panel on Interventional Radiology. ACR Appropriateness Criteria® Radiologic Management of Mesenteric Ischemia: 2022 Update. *J Am Coll Radiol* 2022;19(11S):S433–S444.
27. Garzelli L, Nuzzo A, Copin P, et al. Contrast-enhanced CT for the Diagnosis of Acute Mesenteric Ischemia. *AJR Am J Roentgenol* 2020;215(1):29–38.
28. Expert Panels on Vascular Imaging and Gastrointestinal Imaging; Ginsburg M, Obara P, et al. ACR Appropriateness Criteria® Imaging of Mesenteric Ischemia. *J Am Coll Radiol* 2018;15(11S):S332–S340.
29. Davarpanah AH, Ghamari Khameneh A, Khosravi B, Mir A, Saffar H, Radmard AR. Many faces of acute bowel ischemia: overview of radiologic staging. *Insights Imaging* 2021;12(1):56.
30. Chuong AM, Corno L, Beaussier H, et al. Assessment of Bowel Wall Enhancement for the Diagnosis of Intestinal Ischemia in Patients with Small Bowel Obstruction: Value of Adding Unenhanced CT to Contrast-enhanced CT. *Radiology* 2016;280(1):98–107.
31. Schieda N, Fasih N, Shabana W. Triphasic CT in the diagnosis of acute mesenteric ischaemia. *Eur Radiol* 2013;23(7):1891–1900.
32. Jo PC, Cabral FC, Sahin A, Camacho A, Brook A, Brook OR. Split-bolus single scan CTA for evaluation of mesenteric ischemia. *Abdom Radiol (NY)* 2018;43(6):1368–1378.
33. Lehtimäki TT, Kärkkäinen JM, Saari P, Manninen H, Pääjnen H, Vanninen R. Detecting acute mesenteric ischemia in CT of the acute abdomen is dependent on clinical suspicion: review of 95 consecutive patients. *Eur J Radiol* 2015;84(12):2444–2453.
34. Raman SP, Fishman EK. Computed Tomography Angiography of the Small Bowel and Mesentery. *Radiol Clin North Am* 2016;54(1):87–100.
35. Fulwadhva UP, Wortman JR, Sodickson AD. Use of Dual-Energy CT and Iodine Maps in Evaluation of Bowel Disease. *RadioGraphics* 2016;36(2):393–406.
36. Shaqdan KW, Parakh A, Kambadakone AR, Sahani DV. Role of dual energy CT to improve diagnosis of non-traumatic abdominal vascular emergencies. *Abdom Radiol (NY)* 2019;44(2):406–421.
37. Potretzke TA, Brace CL, Lubner MG, Sampson LA, Willey BJ, Lee FT. Early Small-Bowel Ischemia: Dual-Energy CT Improves Conspicuity Compared with Conventional CT in a Swine Model. *Radiology* 2015;275(1):119–126.
38. Toia GV, Hartman JB, Sahani DV, Mileto A. Dual-Energy CT in the Inpatient Setting. In: Bhosale PR, Marin D, Morgan DE, eds. *Practical Dual-Energy CT Throughout the Body: A Busy Radiologist's Primer*. American Roentgen Ray Society, 2021; 189–195.
39. Lourenco PDM, Rawski R, Mohammed MF, Khosa F, Nicolaou S, McLaughlin P. Dual-Energy CT Iodine Mapping and 40-keV Monoenergetic Applications in the Diagnosis of Acute Bowel Ischemia. *AJR Am J Roentgenol* 2018;211(3):564–570.
40. D'Angelo T, Bucolo GM, Yel I, et al. Dual-energy CT late arterial phase iodine maps for the diagnosis of acute non-occlusive mesenteric ischemia. *Radiol Med* 2024;129(11):1611–1621.

41. Oberparleiter M, Vosschenrich J, Breit HC, et al. Dual-energy CT of acute bowel ischemia: influence on diagnostic accuracy and reader confidence. *Eur Radiol* 2025;35(3):1405–1414.
42. Henes FO, Pickhardt PJ, Herzyk A, et al. CT angiography in the setting of suspected acute mesenteric ischemia: prevalence of ischemic and alternative diagnoses. *Abdom Radiol (NY)* 2017;42(4):1152–1161.
43. Acosta S, Ogren M, Sternby NH, Bergqvist D, Björck M. Clinical implications for the management of acute thromboembolic occlusion of the superior mesenteric artery: autopsy findings in 213 patients. *Ann Surg* 2005;241(3):516–522.
44. Tual A, Garzelli L, Nuzzo A, et al. Strengthening the Description of Superior Mesenteric Artery Occlusions in Acute Mesenteric Ischaemia: Proposition for an Anatomical Classification. *Eur J Vasc Endovasc Surg* 2023;65(6):802–808.
45. Horton KM, Fishman EK. Multidetector CT Angiography in the Diagnosis of Mesenteric Ischemia. *Radiol Clin North Am* 2007;45(2):275–288.
46. Kirkpatrick IDC, Kroeker MA, Greenberg HM. Biphasic CT with Mesenteric CT Angiography in the Evaluation of Acute Mesenteric Ischemia: initial Experience. *Radiology* 2003;229(1):91–98.
47. Yikilmaz A, Karahan OI, Senol S, Tuna IS, Akyildiz HY. Value of multislice computed tomography in the diagnosis of acute mesenteric ischemia. *Eur J Radiol* 2011;80(2):297–302.
48. Reginelli A, Iacobellis F, Berritto D, et al. Mesenteric ischemia: the importance of differential diagnosis for the surgeon. *BMC Surg* 2013;13 Suppl 2(suppl 2):S51.
49. Calame P, Malakhia A, Turco C, Grillet F, Piton G, Delabrousse E. Transmural Bowel Necrosis from Acute Mesenteric Ischemia and Strangulated Small-Bowel Obstruction: Distinctive CT Features. *AJR Am J Roentgenol* 2020;214(1):90–95.
50. Nuzzo A, Maggiori L, Ronot M, et al. Predictive Factors of Intestinal Necrosis in Acute Mesenteric Ischemia: Prospective Study from an Intestinal Stroke Center. *Am J Gastroenterol* 2017;112(4):597–605.
51. Atre ID, Eurboonyanun K, O'Shea A, et al. Predictors of transmural intestinal necrosis in patients presenting with acute mesenteric ischemia on computed tomography. *Abdom Radiol (NY)* 2022;47(5):1636–1643.
52. Garzelli L, Dufay R, Tual A, et al. Predictors of Survival without Intestinal Resection after First-Line Endovascular Revascularization in Patients with Acute Arterial Mesenteric Ischemia. *Radiology* 2024;311(3):e230830.
53. Calame P, Delabrousse É, Ronot M. Letter to the editor: pneumatosis in bowel ischemia—time to change the optics to improve patient care. *Insights Imaging* 2022;13(1):25.
54. Olson MC, Bach CR, Wells ML, et al. Imaging of Bowel Ischemia: an Update, from the AJR Special Series on Emergency Radiology. *AJR Am J Roentgenol* 2023;220(2):173–185.
55. Harnik IG, Brandt LJ. Mesenteric venous thrombosis. *Vasc Med* 2010;15(5):407–418.
56. Russell CE, Wadhera RK, Piazza G. Mesenteric Venous Thrombosis. *Circulation* 2015;131(18):1599–1603.
57. Duran R, Denys AL, Letovanec I, Meuli RA, Schmidt S. Multidetector CT Features of Mesenteric Vein Thrombosis. *RadioGraphics* 2012;32(5):1503–1522.
58. Wang Y, Zhao R, Xia L, Cui YP, Zhou Y, Wu XT. Predictive Risk Factors of Intestinal Necrosis in Patients with Mesenteric Venous Thrombosis: Retrospective Study from a Single Center. *Can J Gastroenterol Hepatol* 2019;2019(1):8906803.
59. Jiang M, Li CL, Pan CQ, et al. Nomogram for predicting transmural bowel infarction in patients with acute superior mesenteric venous thrombosis. *World J Gastroenterol* 2020;26(26):3800–3813.
60. Sun SL, Wang XY, Chu CN, Liu BC, Li QR, Ding WW. Predictors of irreversible intestinal resection in patients with acute mesenteric venous thrombosis. *World J Gastroenterol* 2020;26(25):3625–3637.
61. Calame P, Winiszewski H, Doussot A, et al. Evaluating the Risk of Irreversible Intestinal Necrosis among Critically Ill Patients with Nonocclusive Mesenteric Ischemia. *Am J Gastroenterol* 2021;116(7):1506–1513.
62. Rossi UG, Petrocelli F, Seitun S, Ferro C. Nonocclusive Mesenteric Ischemia in a Dialysis Patient with Extensive Vascular Calcification. *Am J Kidney Dis* 2012;60(5):843–846.
63. Pérez-García C, de Miguel Campos E, Fernández Gonzalo A, et al. Non-occlusive mesenteric ischaemia: CT findings, clinical outcomes and assessment of the diameter of the superior mesenteric artery. *Br J Radiol* 2018;91(1081):20170492.
64. Wilcox MG, Howard TJ, Plaskon LA, Unthank JL, Madura JA. Current theories of pathogenesis and treatment of nonocclusive mesenteric ischemia. *Dig Dis Sci* 1995;40(4):709–716.
65. Nakamura Y, Kondo S, Narita K, et al. Understanding CT imaging findings based on the underlying pathophysiology in patients with small bowel ischemia. *Jpn J Radiol* 2023;41(4):353–366.
66. Verdot P, Calame P, Winiszewski H, et al. Diagnostic performance of CT for the detection of transmural bowel necrosis in non-occlusive mesenteric ischemia. *Eur Radiol* 2021;31(9):6835–6845.
67. Barrett T, Upponi S, Benaglia T, Tasker AD. Multidetector CT findings in patients with mesenteric ischaemia following cardiopulmonary bypass surgery. *Br J Radiol* 2013;86(1030):20130277.
68. Paulson EK, Thompson WM. Review of small-bowel obstruction: the diagnosis and when to worry. *Radiology* 2015;275(2):332–342.
69. Li BQ, Qi WJ, Yuan M, et al. Increased Attenuation of Intestinal Contents at CT Indicates Bowel Necrosis in Closed-Loop Small Bowel Obstruction. *Radiology* 2024;310(2):e231710.
70. Millet I, Taourel P, Ruyar A, Molinari N. Value of CT findings to predict surgical ischemia in small bowel obstruction: a systematic review and meta-analysis. *Eur Radiol* 2015;25(6):1823–1835.
71. Olson MC, Fletcher JG, Nagpal P, Froemming AT, Khandelwal A. Mesenteric ischemia: what the radiologist needs to know. *Cardiovasc Diagn Ther* 2019;9(suppl 1):S74–S87.
72. Garzelli L, Nuzzo A, Hamon A, et al. Reperfusion injury on computed tomography following endovascular revascularization of acute mesenteric ischemia: prevalence, risk factors, and patient outcome. *Insights Imaging* 2022;13(1):194.
73. Kernagis LY, Levine MS, Jacobs JE. Pneumatosis Intestinalis in Patients with Ischemia: Correlation of CT Findings with Viability of the Bowel. *AJR Am J Roentgenol* 2003;180(3):733–736.
74. Wiesner W, Mortelé KJ, Glickman JN, Ji H, Ros PR. Pneumatosis Intestinalis and Portomesenteric Venous Gas in Intestinal Ischemia. *AJR Am J Roentgenol* 2001;177(6):1319–1323.
75. Duron VP, Rutigliano S, Machan JT, Dupuy DE, Mazzaglia PJ. Computed Tomographic Diagnosis of Pneumatosis Intestinalis: Clinical Measures Predictive of the Need for Surgical Intervention. *Arch Surg* 2011;146(5):506–510.
76. Cokkinis AJ. Mesenteric Vascular Occlusion: Supplemented by an Appendix of 76 Original Cases. Bailliere, Tindall & Cox, 1926.